# EXTERNAL DISEASES OF THE EYE

# ATLAS

OF

# EXTERNAL DISEASES OF THE EYE

FOR

# PHYSICIANS AND STUDENTS

BY


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ONLY AUTHORIZED ENGLISH TRANSLATION

BY


## P. W. SHEDD, M.D.

NEW YORK


WITH 84 ILLUSTRATIONS IN COLOR FROM WAX MODELS PRINTED ON 54 PLATES WITH EXPLANATORY TEXT. THE ILLUSTRATIONS ARE FROM MODELS IN THE PATHOPLASTIC INSTITUTE IN BERLIN

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DEDICATED

TO

MY DEAR COLLEAGUE

DR. THEODOR AXENFELD

Professor of Ophthalmology and Chief
of the Ophthalmic Clinic in the
University of Freiburg

In Remembrance of true and unbroken
friendship since the days of
student life

# Preface.

For years, in conjunction with the sculptor, F. Kolbow, I have endeavored to perpetuate in wax models the appearance of important external diseases of the eye. Because of the delicacy and sensitivity of ophthalmic tissues, the difficulties to overcome were, naturally, very great, and only after much experiment were we able to discover the correct method.

The request of the publishers, Urban & Schwarzenberg, to prepare an Atlas of the most important external diseases of the eye was acceded to only when I was convinced that modern art could produce pictures of distinctly better technic than those hitherto published.

The plates were obtained by making, after a special method, a mask of the living subject, which was then filled with wax. From the wax models photographic reproduction in four colors was made.

We thus gain two advantages, *viz.,* we have before us, not a schematic representation, but the actual case as it appeared in the University Ophthalmic Clinic of the Berlin Charité Hospital of which I am chief, and, so to speak, no man's hand has touched it. Furthermore, by means of the models, we get a pictorial plasticity unattainable by the most accomplished draughtsman.

Although the work appears under my name, I am fully conscious that only a portion thereof is actually mine.

To the skill, powers of observation and zealous par-

ticipation of Sculptor Kolbow (Berlin), success in obtaining the beautiful plates is largely due. He is, probably, surpassed by no one to-day in the fabrication of wax models.

A number of the false eyes inserted into the models simulate the pathologic aspect of the organ, and were prepared in accordance with my specifications by the optical firm of Müller Bros. (Wiesbaden) and by Hans Hüning (Berlin).

The drawings were made by the expert scientific draughtsman, H. Helbig.

The execution of the plates in color was undertaken by the firm of Dr. Selle & Co. The difficulties here were so great that at first many of the plates were necessarily rejected. Finally, I may mention with commendation the willingness of the firm to make all alterations and corrections, and that, too, with no diminution of its productive ability.

The printing of the plates was most carefully and satisfactorily accomplished by the firm of Döring & Huning (Berlin).

Figs. 4 and 5 were taken from Bockenheimer's Atlas of Surgical Diseases, and Fig. 8 from the Atlas of Skin Diseases by Jacobi, both of which works are published by the same house.

Last, but not least, an expression of my gratitude and content is due the publishers, Urban & Schwarzenberg, who, notwithstanding the enormous expense of the undertaking, have invariably sought to conjoin in the work the best offered by modern science and technic, neglecting no method or experiment necessary to such accomplishment.

We hope for a substantiation of our aim in preparing this volume.

Prof. R. Greeff.

Berlin, February, 1909.

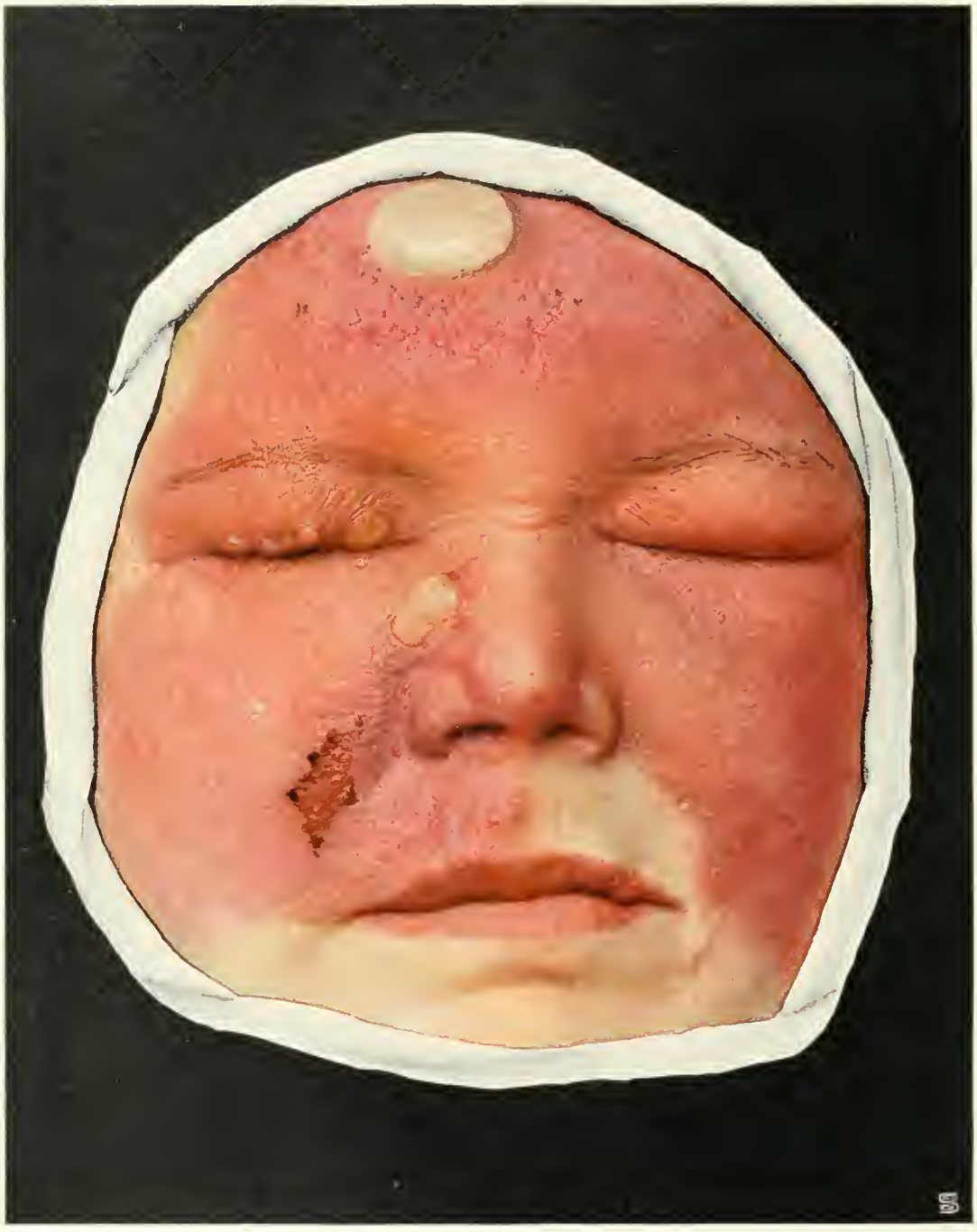

Fig. 1.
Erysipelas Faciei — Oedema palpebrarum

# Erysipelas Faciei, Oedema Palpebrarum.

Plate I., Fig. 1.

Facial erysipelas, also called St. Anthony's fire (German: Gesichtsrose, Rotlauf), follows, commonly, some slight injury of the skin (vesicle, bleb, eczema, excoriations, rhagades), which serves as a point of entry for the *streptococcus erysipelatis*. With high fever, sometimes with chills, there develops a sharply-contoured, glistening red swelling of the skin, which extends rapidly, and at last, usually involves the entire face. Within a short time the epiderm is raised up in few or many blebs.

The eyelids present a markedly reddened skin; upon their margins the blebs are particularly apt to develop (Fig. 1, right eye); and there is great tumefaction, so that, as a rule, the eyes cannot be opened. The extreme inflammatory palpebral edema is due to the loose connection here betwixt the skin and the underlying tissues, to the absence of subcutaneous fat, and to the fact that there is present abundant space for the extension of a tumefactive process (*Cf. infra*: Edema).

Commonly, the temperature gradually falls after a few days; the blebs or vesicles break, and, with retrogression of the redness and swelling, there begin exfoliation and pigmentation of the affected areas.

The **diagnosis** is easily made. The simultaneous appearance of fever with rubescent and glistening skin renders error difficult.

The **prognosis** is, in general, good, but should be carefully guarded in expression. When there is high fever and exhaustion, indicative of a general infection, death may follow. In very serious cases palpebral gangrene may develop as an unpleasant complication (*vide,* p. 11).

It is to be remembered that in convalescent cases sudden relapses are not infrequent. The disease confers no immunity; on the contrary, one attack predisposes to another.

**Therapy** is local in character and consists in the application of unguents which render the tense skin more supple and also have an excellent subjective effect. Neutral unguents (boracic acid, resorcin, etc.) are best. Others prefer cold applications (wet cloths). Where there is menacing tension of the lids and extreme pain, scarification of the skin may be considered as a last resort (*vide* Gangrene, p. 11).

General treatment, *i. e.*, rest in bed, fever-diet, etc., needs no discussion.

Erysipelas is extremely infectious, and requires, therefore, the most perfect isolation possible.

**Edema of the eyelids** is not, commonly, a nosologic entity.

As we have already remarked, the derm of the lid is not intimately bound to the subjacent tissues, so that fluid exudates find abundant space beneath the palpebral skin and, therefore, are often apt to extend to a considerable distance. This is best seen in palpebral hemorrhage (*vide* Fig. 2).

Inflammatory edemas may often arise from slight irritations (*e. g.,* bee-stings, fly-bites) and spread until the lids appear like bags distended with water, the eye being completely occluded. Palpebral edemas,

then, are mostly secondary phenomena, and are also noted in cases of abscess, chalazion, hordeolum, dacrocystitis, orbital phlegmon, panophthalmia, etc.

Furthermore, it is well-known that anasarca, with simultaneous swelling of the ankle, is also apt to develop palpebral edema (renal disease).

A doughy edema of the lids is found in trichinosis, the trichinæ having a predilection for the orbital muscle.

# Hæmorrhagia Subdermalis et Subconjunctivalis

Plate II., Fig. 2.

Palpebral sugillation or suffusion, *i. e.*, hæmorrhagia subdermalis, is a very striking phenomenon. Because of the spongy tissue beneath the skin, the blood extends easily and far. The red tint soon changes to a reddish-blue and then becomes blue-black (the well-known black eye resulting from a blow). The hemorrhage is, commonly, sharply limited at the orbital margin, for here the skin is firmly attached to the bone by tense connective tissue. The skin about the root of the nose is, on the contrary, loosely bound to the subjacent tissue. Hence, the hemorrhage beneath the skin of one eye may extend under the skin of the nasal bridge and appear, with correspondent coloration, beneath the skin of the uninjured optic. It frequently happens that, after operation on one eye, hemorrhagic discoloration develops in both. In such cases one should not be misled to the conclusion that both eyes had suffered injury.

Sugillation of the lids is particularly a sequela of trauma, notably that due to blows with blunt objects (fist, club, etc.); also of major operations, such as enucleation, where the wound is deep, whilst in cuts of palpebral tissue it is rarely observed, for the blood has abundant exit.

Spontaneous bleeding may also occur, for the vascular channels, from lack of supporting tissue, are

Fig. 2.
Haemorrhagia subdermalis et subconjunctivalis.

easily ruptured, as during violent exertion, by crushing, sneezing, coughing, etc.

Palpebral sugillation is of special significance in diagnosing fracture of the base of the skull. In this grave injury the blood often travels from the seat of fracture forwards along the floor of the orbit, appearing, usually after some lapse of time, beneath the conjunctiva (*vide infra*) and the skin of the lower lid, particularly in the region of the inner canthus.

The discolored skin permits instant diagnosis, and only the cause of the hemorrhage demands further investigation.

The **prognosis** is commonly favorable. The discoloration gradually takes on a greenish hue, and in most cases the blood is resorbed after a few weeks. Rarely, the effusion passes into suppuration, thus forming a palpebral abscess.

**Therapy.** Cool applications.

Subconjunctival hemorrhage develops even more easily, for here, likewise, there is very loose attachment to underlying tissues, and we have: *haemorrhagia subconjunctivalis, hyphaema conjunctivae* or, briefly, *hyposphagma.*

In youth it almost invariably accompanies pertussis and may also be caused by immoderate coughing, pressure or strangling in children. In older individuals it is indicative of vascular fragility, of arteriosclerosis, and often accompanies contracted kidney.

Conjunctival hemorrhages have, therefore, weighty symptomatic significance.

The **diagnosis** is not difficult: the uniform, superficial reddening, if once seen, will not be confused with an inflammation of the conjunctiva, where the individual dilated blood-vessels are easily distinguished.

Local **therapy** is of little value. The striking phenomenon of a subconjunctival hemorrhage usually terrifies the patient or those about him, but they may be easily calmed, for the eye is never damaged. However, lead-water or cold compresses should be applied. The constitutional cause of the hemorrhage is to be sought and treated. The phenomenon is frequently a prodrome of cerebral apoplexy.

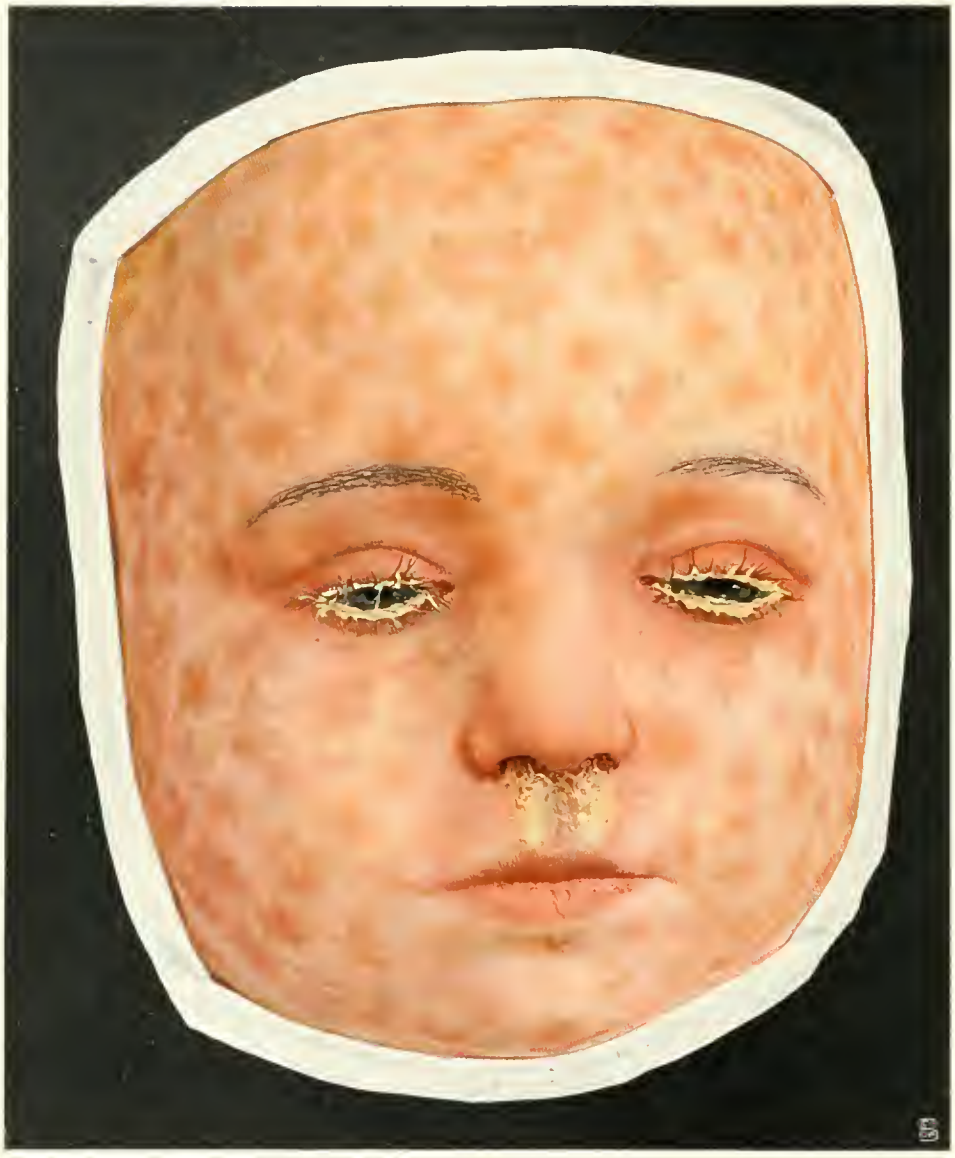

Fig. 3.
Morbilli. Blepharo-Conjunctivitis exanthematica.

# Morbilli—Conjunctivitis Exanthematica.

Plate III., Fig. 3.

With the efflorescence of the exanthem in the various acute general infections, but notably so in measles, characteristic catarrhal phenomena commonly appear in the form of more or less violent conjunctivitis and blepharitis with redness, photophobia, and secretion, as well as catarrhal conditions in the nose and upper air-passages. These catarrhal conditions may even precede the exanthematous efflorescence by some days, the secretion consisting either of increased lacrimal fluid or, not rarely, of a mucous or purulent exudate drying into scales and crusts along the margin of the lid (*vide* Fig. 3). Croupous membranes seldom develop.

**Prognosis.** In measles the acute conjunctival catarrh always present is not to be slighted Though spontaneously disappearing, in most cases, after 2-3 weeks, it may, if neglected, lead to a redness and sensitivity of the blepharo-conjunctival tissues, annoying the patient for years or during his entire life. Serious complications such as a blennorrhoic or diphtheritic conjunctivitis or corneal infiltration or a secondary iritis are not impossible.

**Therapy.** As with measles in general, cleanliness plays a chief role in the ophthalmic treatment. The lids are to be bathed carefully with lukewarm

boracic water and the dried exudate softened and removed. In most cases, this will suffice. Where lacrimation is more abundant, one drop of a slightly astringent collyrium (acid. tannicum 1%, resorcin 1%, zinc sulfate ¼%) may be used daily. With purulent secretion or the formation of membrane, irrigation with a $^1/_{10}$ or $^1/_4$% solution of silver nitrate is indicated. Where such conditions develop, the globe should be closely watched for pericorneal injection (atropin) or corneal infiltration.

The room is to be kept moderately darkened and the child not exposed to ordinary light until all ophthalmic irritation has subsided.

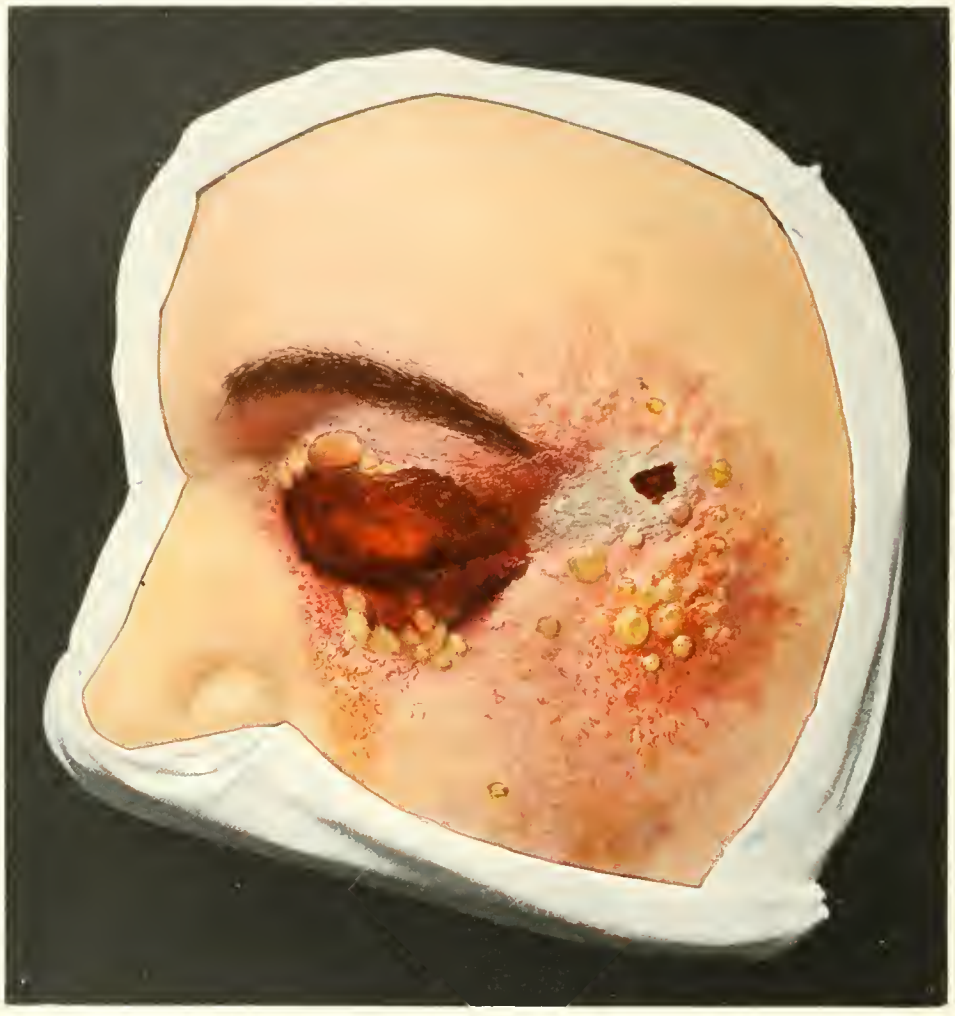

Fig. 4.
Pustula maligna — Anthrax.

# Pustula Maligna—Anthrax.

Plate IV., Fig. 4.

The anthrax pustule or *pustula maligna* not infrequently appears upon the eyelid. Man is inoculated with the bacillus anthracis from diseased animals by wiping, rubbing or scratching the eyes with the hand, and malignant pustule is therefore found in individuals handling animal products — cattle dealers, butchers, tanners, dealers in leather or furs. The affection often begins with a vesicle on the margin of the lid filled with yellow turbid or bloody matter. There is also a violent inflammatory edema of the lid and tense infiltration of the skin. Soon swelling of the preauricular and submaxillary glands and fever develop, followed by rupture of the pustule which becomes covered by a scab. The surrounding skin then turns a grayish color, indicative of commencing necrosis.

**Diagnosis.** Similar vesicular formation may also be found in phlegmonous inflammations, carbuncle, and in glanders. Bacteriologic discovery of the specific bacilli (non-mobile rods with square-cut ends, often in long chains) renders the diagnosis certain.

The **prognosis** is extremely bad, for the lids usually become necrosed, and the case terminates in death.

Formerly, the **therapy** was surgical: incision, currettage, or a Paquelin cauterization. We have learned, however, that the less the site of infection is

disturbed, the less danger there is of bacterial entrance into the blood stream. Bearing this in mind, the best treatment is the application of unguents or aseptic compresses. The scabs and necrotic tissues are left for gradual and spontaneous desquamation. If the infection do not terminate fatally, plastic surgery is indicated.

Fig. 4 shows a malignant pustule of the lid, whose reproduction in this atlas was kindly permitted by Prof. Dr. Bockenheimer. The case is one of external anthrax infection in a laborer employed in a tannery, and developed from a slight scratch in the skin of the cheek. At first, a red nodule appeared, then several vesicles filled with a yellow fluid of bacillary content. There was widespread carbunculoid infiltration, marked edema of the lids and an erysipelatoid reddening of the entire cheek. Soon after rupture of the vesicle a scab formed at the site of infection, with an areola of grayish skin gradually passing into necrosis. The process, with marked systemic involvement, fever, chills, delirium, then extended to the eyelids, which, because of the enormous tumefaction, could no longer be opened even by force. Pustule after pustule developed, with correspondent gangrene of the skin after their rupture. The entire half of the face was protected by an unguental application.

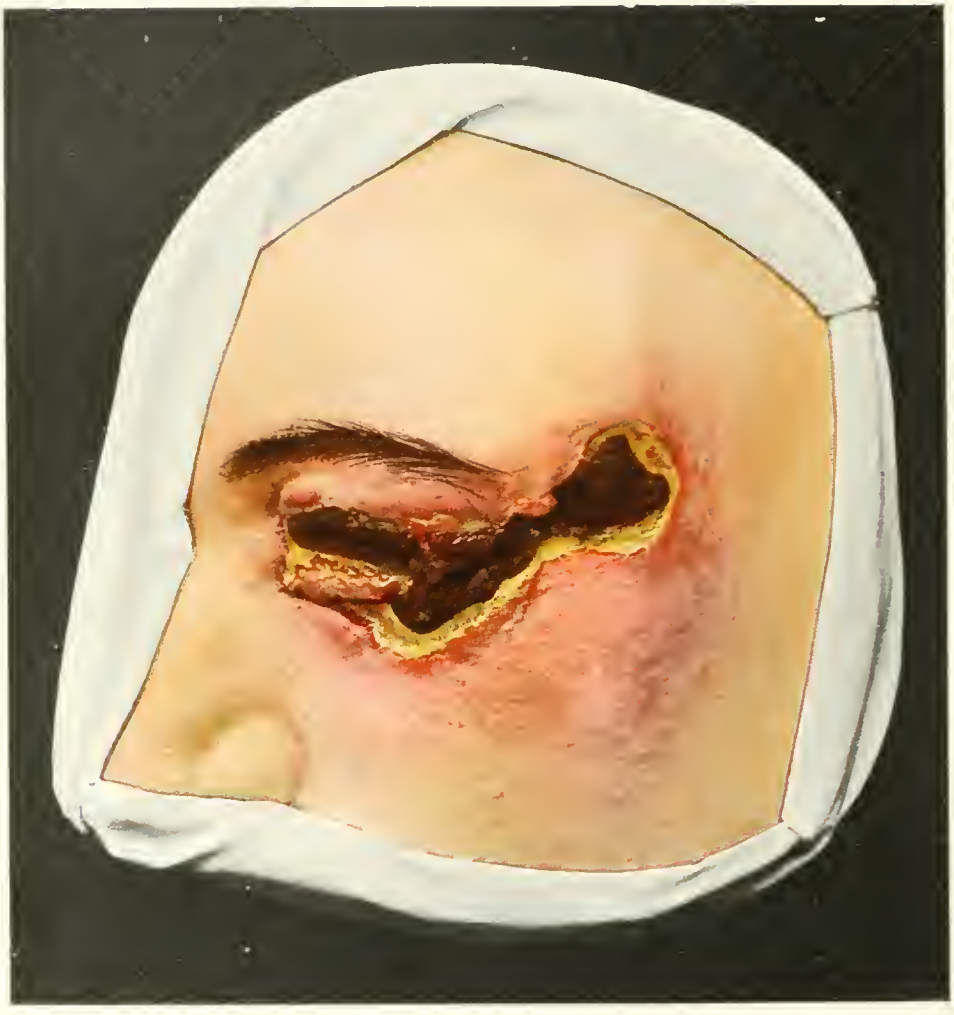

Fig. 5.
Gangraena palpebrarum— Anthrax.

# Gangræna Palpebrarum. Anthrax.

Plate V., Fig. 5.

The delicate texture of the palpebral skin, its thin corium, the loose subcutaneous tissues with their large lymph spaces and the richness of the vascular supply, permit easy extension of a malignant inflammation and trophic disturbance of tissue. We are speaking of gangrene when the breaking-down of tissue elements occurs with decomposition and putrefaction. In such case, we find in the palpebral region a circumscribed, fetid necrosis surrounded by a zone of inflammatory reaction.

According to Römer, to whom we are indebted for a study of the subject, gangrene may develop endogeously, *i. e.,* by metastasis, or ectogenously, *i. e.,* from some local disturbance.

I. *The Endogenous Form.* Metastatic gangrene of the lids develops, but not often, in severe general diseases, particularly typhoid, measles, scarlatina. Even in 1794, Himly reported that in grave typhoid the eyelids became blue and sphacelated within a few hours. Fieuzal gives three cases of palpebral gangrene during measles, and similar cases are recorded by Knies and Randall.

Partial gangrene of the eyelid in scarlatina is described by St. Martin and Jackson, whilst numerous palpebral abscesses have been observed in influenza.

In pyemia and sepsis, gangrene of the lid is caused

by infectious emboli, and it has also been attributed to diabetes and alcoholism.

II. *The Ectogenous Form.* Here the necrosis may proceed from foci of inflammation in the neighborhood of the eye, or develop primarily in the palpebral tissue.

Secondary necroses of the lid are observed most frequently as complications of facial erysipelas. The minutest infected wound often plays a role here, and more extensive injuries are not rarely etiologic. Schmidt-Rimpler reports a case where, after a blow from a twig upon the malar bone, tumefaction of the eyelid developed, and five days later the palpebral tissue was transformed into an ulcer full of necrotic shreds.

Among the primary affections of the lid where gangrene is possible, anthrax is pre-eminent. In Grossmann's case, we are, doubtless, dealing with *pustula maligna.* In a broom-maker a pustule as large as a pea developed, with high fever, on the skin of the upper eyelid, whence a brawny edema spread, reaching even to the thorax. By the third day the skin of the entire lid had become transformed into a black crust. Cure followed, but with extreme ectropion.

For **therapy** consult page 9.

In Fig. 5, we have the case of Prof. Bockenheimer some weeks after the infection. The extensive dermal gangrene, cognizable by the black discoloration and leathern consistency, is already delimited by a zone of pus and unctuous granulation tissue from the adjacent non-gangrenous skin which, however, is slightly reddened and gives evidence of inflammatory infiltration. But the necrosis is still firmly attached to the subjacent tissue. Its forcible removal by the knife or a clumsy extirpation would result only in renascence of the infection. Gradual desquamation was, there-

fore, attained by compresses wet with hydrogen peroxid and boric acid solution and by applications of ointments. In this case, after the loosening of necrotic tissues and subsidence of inflammatory processes in the circumjacent skin, the somewhat marked defect caused by the loss of the entire upper lid was plastically corrected by a pediculated flap of skin from the surrounding healthy tissue. Despite the unfavorable prognosis in facial anthrax and the severity of the local process, the case was cured.

# Herpes Facialis.

Plate VI., Fig. 6; Plate VII., Figs. 7-8.

Herpes zoster is an exanthematous disease of the skin, simulating an infection. In the territory supplied by some particulate nerve a vesicular eruption occurs with febrile phenomena and general weakness. The number of vesicles is extremely variable in different cases. Spring and autumn are the seasons of election.

The vesicle contains at first a clear watery fluid which soon becomes turbid and purulent. It then ruptures and the resulting *ulcus* crusts over.

After the ulcers heal, permanent scars remain, and as a rule, the individual is thenceforth immune.

Of the cranial nerves the trigeminus in all its branches is most often affected and we have a *herpes zoster ophthalmicus,* usually along the course of the first branch of the nerve. In such case the vesicles are found on the upper lid, the forehead to the hairy margin of the scalp, and on the nose, although, because of the almost invariable onesidedness of the affection, they are plainly delimited by the median line of the face (*vide* Plate VII., Fig. 7). In Fig. 8, Plate VIII., there are uncommonly numerous, ruptured vesicles covered with crusts which penetrate deeply into the corium. This latter case is borrowed from Jacobi's Atlas of Skin Diseases.

If the 2nd branch of the trigeminus is affected, the vesicles are located upon the lower lid in the superior maxillary or malar region (Fig. 6, Plate VI).

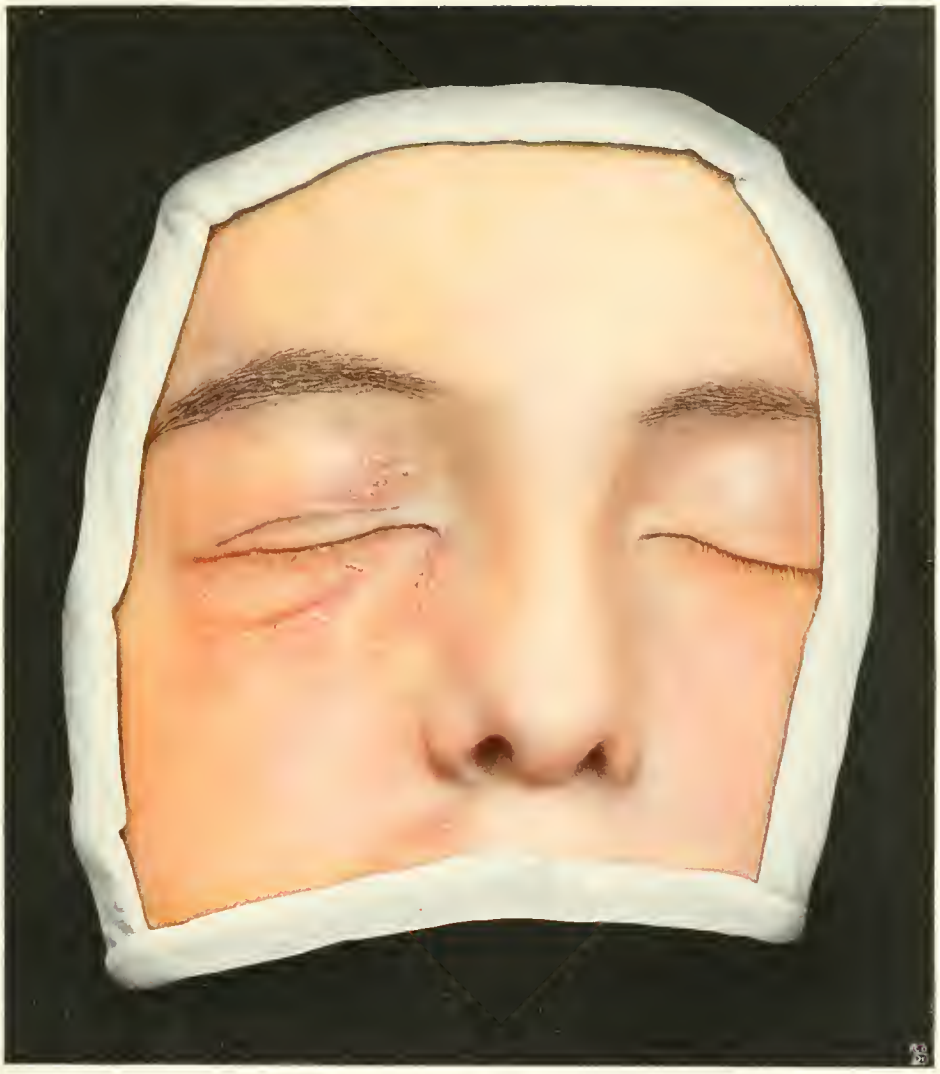

Fig. 6.
Herpes facialis

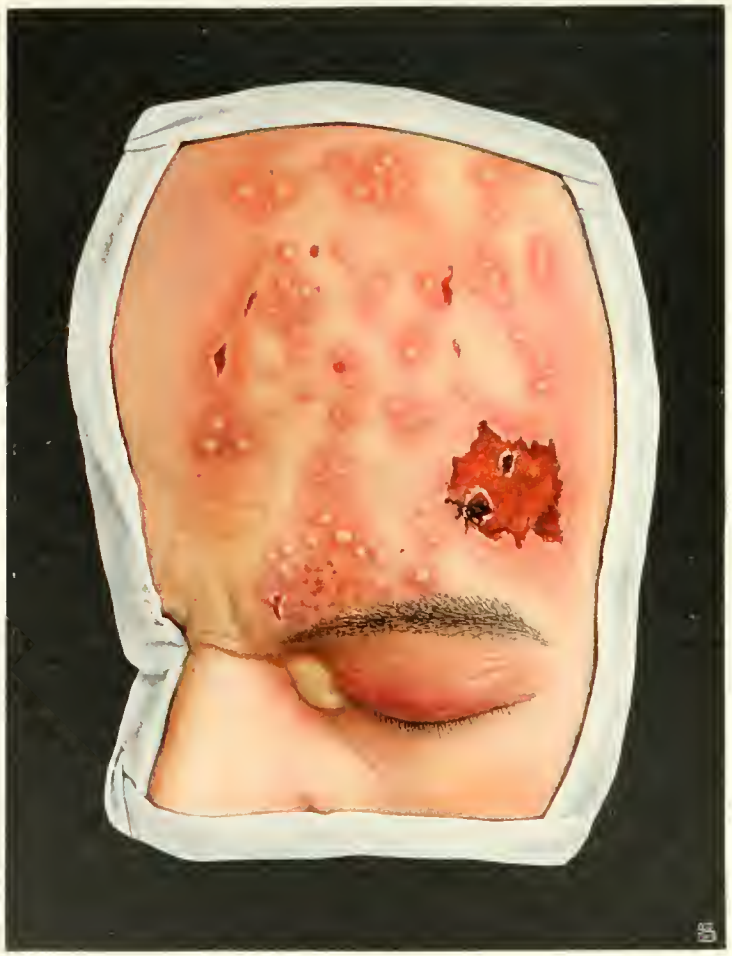

Fig. 7
Herpes zoster ohpthalmicus

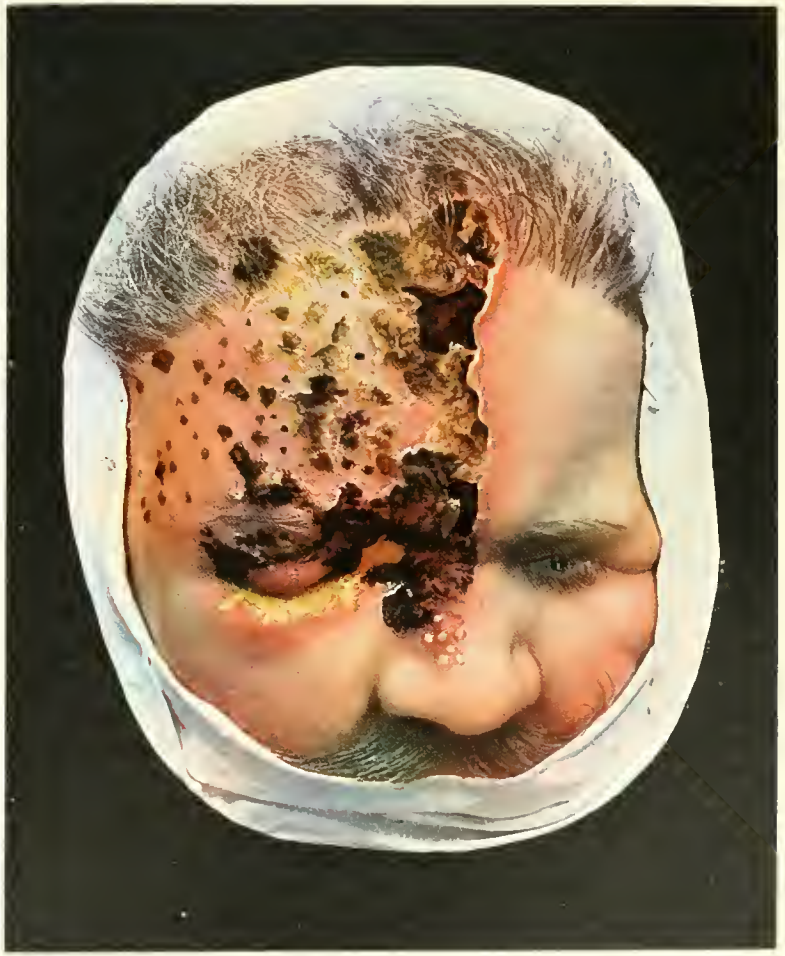

Fig. 8.
Herpes zoster gangraenosus.

Rebman Company, New York

Not infrequently the skin disease is accompanied by an eruption of vesicles on the cornea, a grave complication.

*Herpes zoster ophthalmicus* is due to an inflammatory involvement of the trigeminus, either of the Gasserian ganglion, of the ciliary ganglion or of the nerve in its peripheral course.

**Diagnosis** of *herpes zoster ophthalmicus* is easy because of the distribution of the vesicles within a certain neural territory, the one-sidedness, and the synchronous febrile development. It is differentiated from simple herpes febrilis (labialis) by the size of the vesicles. In simple herpes the epiderm only is vesiculated whilst in herpes zoster the ulcer sinks deep into the substance of the corium and a cicatrix remains after recovery.

**Prognosis** is positively favorable. The scars left may later cause some annoyance.

**Therapy.** Internally, salicyl preparations are called for; for neuralgic pains, quinine, antipyrin or phenacetin. The affected areas are best powdered with rice-starch, lycopodium, etc., which dry up the vesicles. When crusted over, the ulcerated surface heals.

# Variola Vaccina.

Plate VIII., Figs. 9-10.

Vaccinal ophthalmia develops from infection of the eye with the lymph, generally by direct transmission by the finger from the vaccination into the optic; but also from dried lymph on bandages or the handkerchief.

On the eyelids the eruption is usually found along the intermarginal portions. From small, superficial vesicles there develop, with marked inflammatory symptoms, chemosis, palpebral edema, and large, flat ulcers of a diphtheritic appearance. After 8 to 12 days healing begins with complete restitutio ad integrum in 2 to 3 weeks.

The extreme brevity of the incubation stadium may in many cases be reduced to 3 to 4 days.

The variola may also be localized upon the conjunctiva or the cornea.

The **diagnosis** of vaccinal infection of the eye can offer little real difficulty, if we consider the morbidity (Schirmer). Differentiation from *variola vera,* where the pustules cover the entire surface of the body and grave constitutional symptoms are present, is easy.

The *ulcus durum* (chancre) does not, as a rule, cause such marked inflammatory phenomena in the region affected.

Confusion with diphtheritic conditions is possible, but in the rarer diphtheritic ulceration on the margin

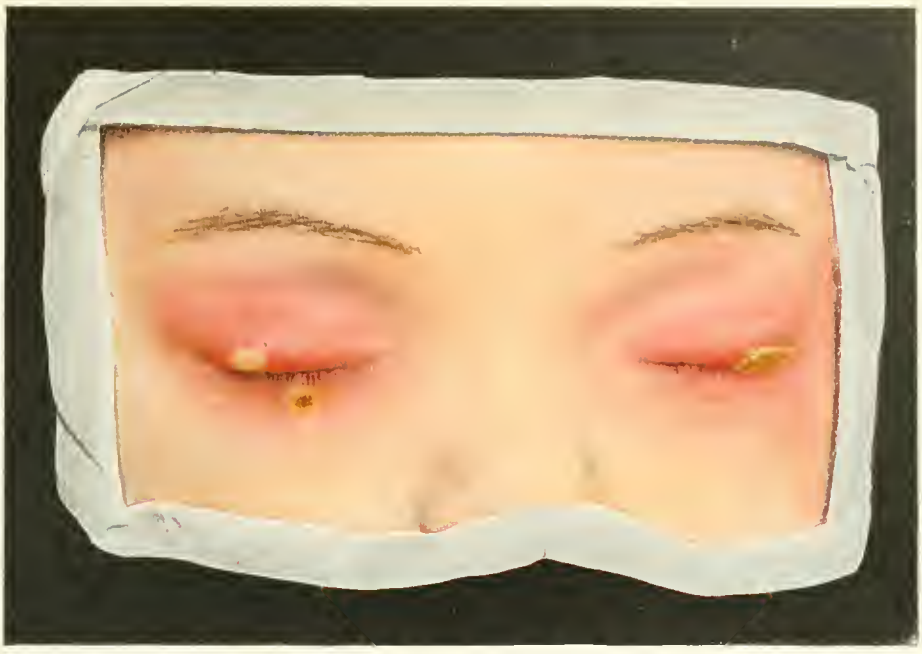

Fig. 9. Variola vaccina I.

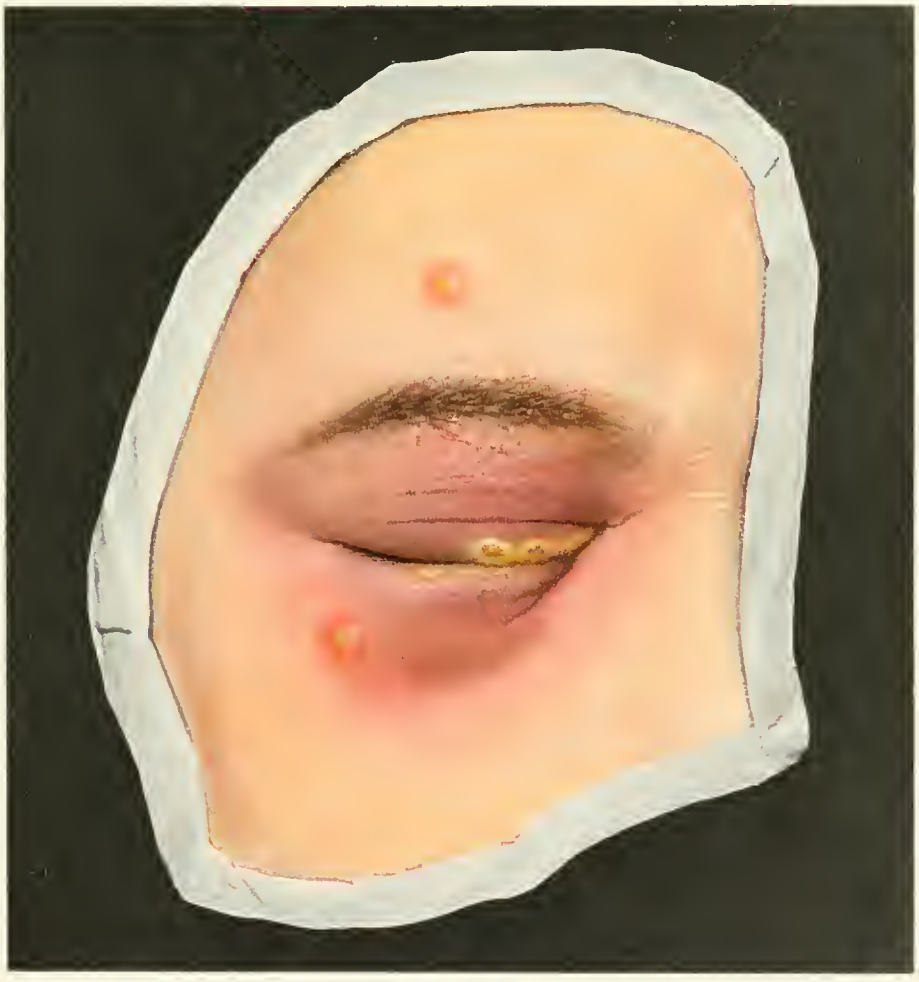

Fig. 10. Variola vaccina II.

of the lid, the whitish membrane covering the conjunctiva is seldom lacking; furthermore, in the vaccinal ulceration removal of the membrane leaves a clear red base whilst in diphtheria the base of the ulceration is dirty.

**Therapy.** Treatment should be as unobtrusive as possible: the eye should be kept clean and the ulcer covered with some unguent. Cauterizing applications are dangerous.

Keratitis profunda or an ulcus corneæ will develop severe complications, and treatment should be that adapted to these affections.

Prophylactically, the family should be warned that the vaccinal virus may be transmitted to the eye by rubbing, etc.

# Ulcus Durum.

Plate IX., Fig. 11.

The margin of the eyelid is not rarely the seat of primary infection, usually due to kissing or to transmission by the finger. The indurations seldom develop on the outer skin but are almost invariably intermarginal or in the canthi or upon the conjunctiva tarsi. The reason therefor is plain. The cutis of the lid is not especially permeable, but the delicate textures of the canthi where derm changes to mucosa, where the glands of the ciliary follicles and the Meibomian glands exude their contents offer facile inoculation of the virus. The pre-auricular and other glands are often so swollen that a diagnosis of mumps might be possible.

The affection begins with a swelling at whose apex a slight excoriation increasing in depth develops, so that finally an ulcer (rarely deep) with well-defined, indurated margins is present.

**Diagnosis** of the extragenital sclerosis in the early stadia is extremely difficult, yet error would be fateful where the lesion is facial, particularly if on the eye or the organs protecting it, because of later possible diminution in function. In differentiation from similar morbid syndromes: hordeolum, chalazion, vaccine pustule, lupus, tuberculosis, diphtheria, chancroid and gumma, the most dependable pathognomic indication is the frequently marked, but indolent, swelling of neighboring glands, in particulate, the

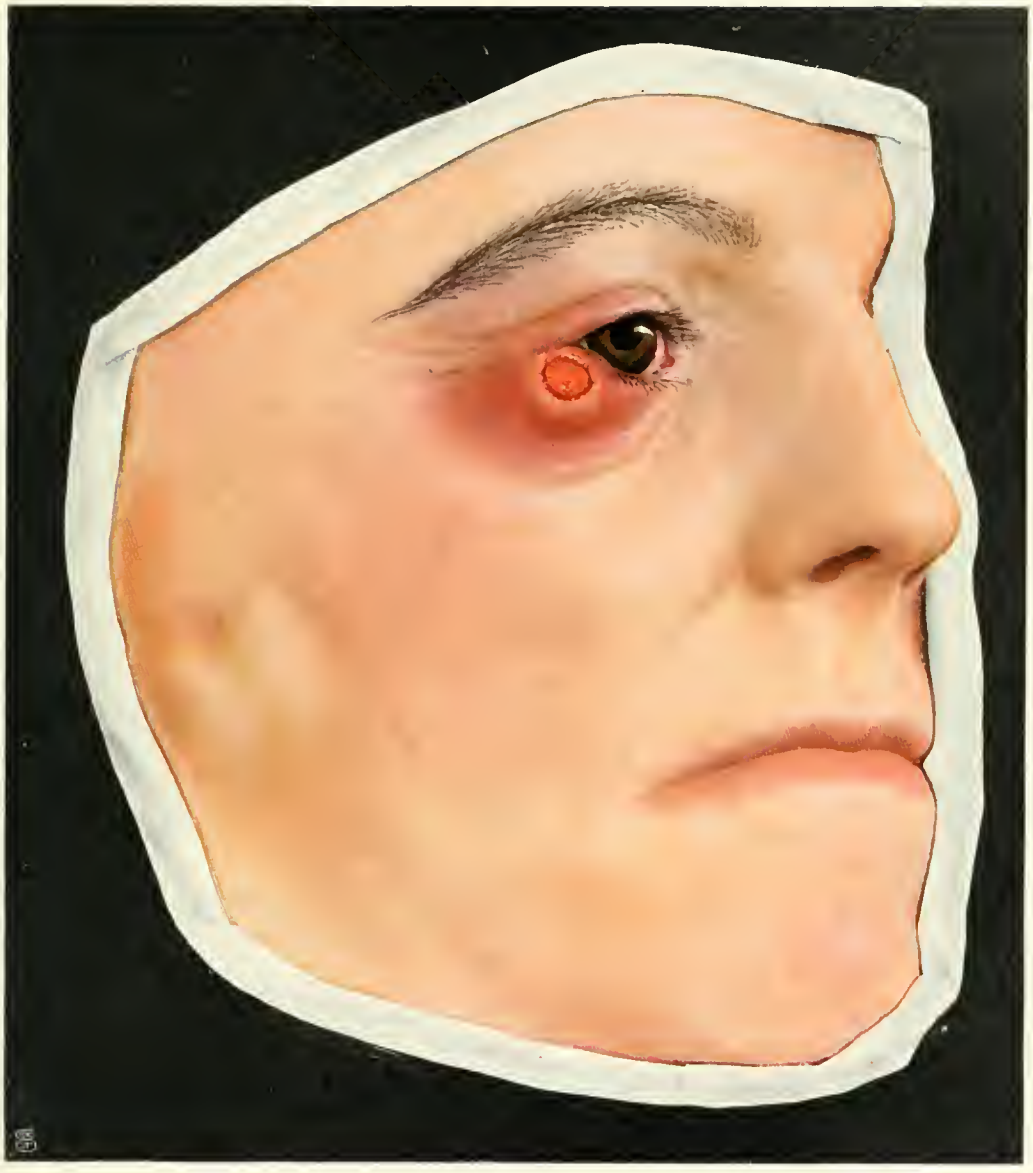

Fig. 11.
Ulcus durum palpebrae. Syphilite. Primary affection.

pre-auricular glands. Not rarely, however, only the appearance of secondary phenomena and the result of mercurial treatment permit a decision.

Kowalewski was the first to render a diagnosis by demonstrating the spirochæta pallida in a palpebral ulcer.

**Therapy** consists in local cleanliness by irrigation with weak antiseptic solutions followed by the application of some indifferent salve. Dusting with iodoform may greatly augment the inflammation without other benefit. The diagnosis once certain, there is no reason for delaying the constitutional treatment.

# Xanthelasma.

Plate X., Fig. 12.

Xanthelasma is a flat, straw- or sulfur-colored tumor located in the palpebral skin, and generally multiple in both upper and lower lid in the region of the inner canthus. Often a tendency is noted to develop symmetric figures on the two eyes. These tumors are found only in elderly adults and more often in women past the menopause. They grow very slowly and injure only cosmetically. When extirpated they rarely return.

Dermatologists differentiate a X. planum and a X. tuberosum, the first of which is found only upon the lids.

Anatomically they are composed of the so-called xanthoma-cells (Touton), *i. e.* hypertrophied connective tissue cells, lying in nests and filled with fat-droplets. These nests are separated from one another by walls of connective tissue. Giant-cells are not infrequently found in them.

**Therapy** is entirely cosmetic and consists of easily executed removal.

**Prognosis.** The tumors are absolutely benign, causing only disfiguration.

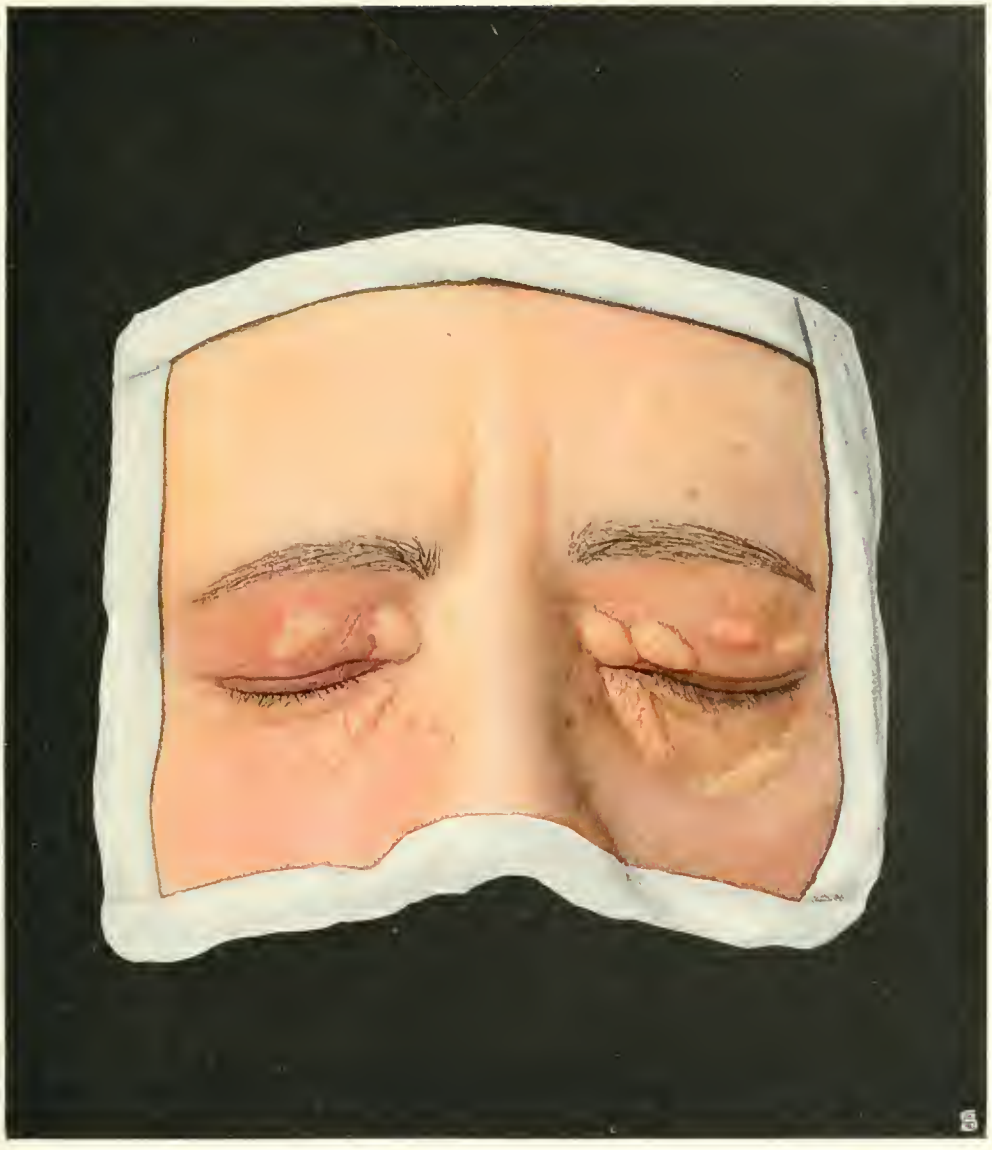

Fig. 12.
Xanthelasma.

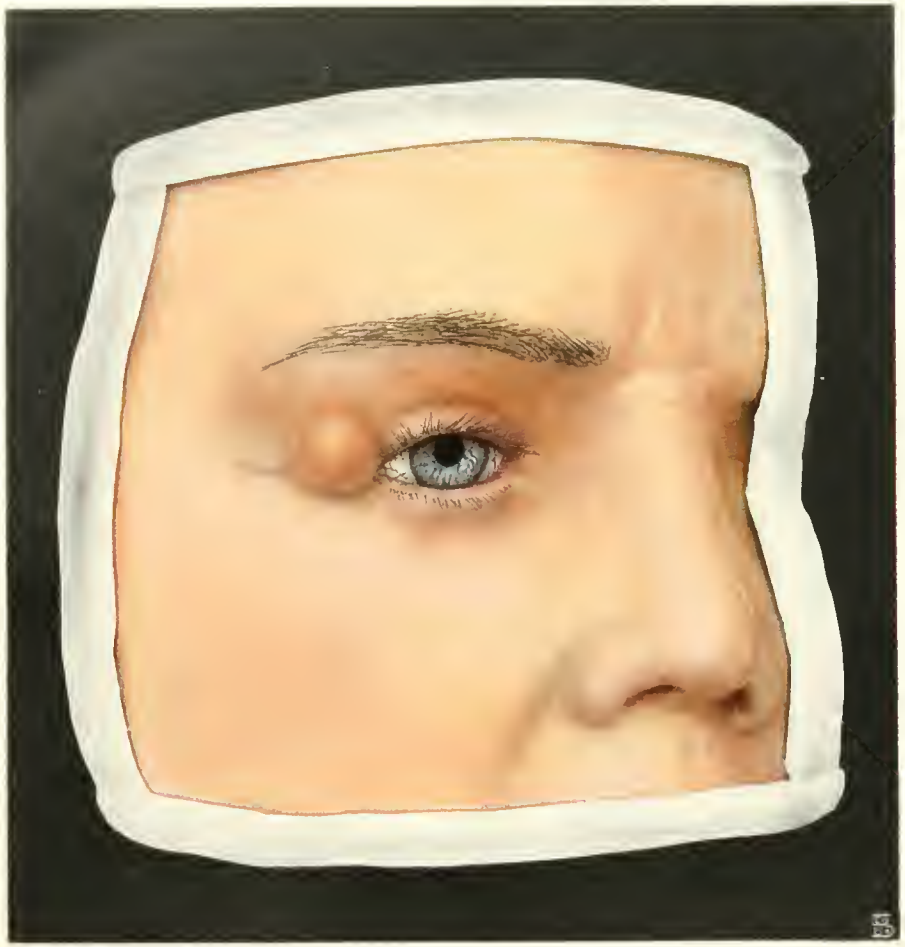

Fig. 13.
Atheromcyste on lid rim.

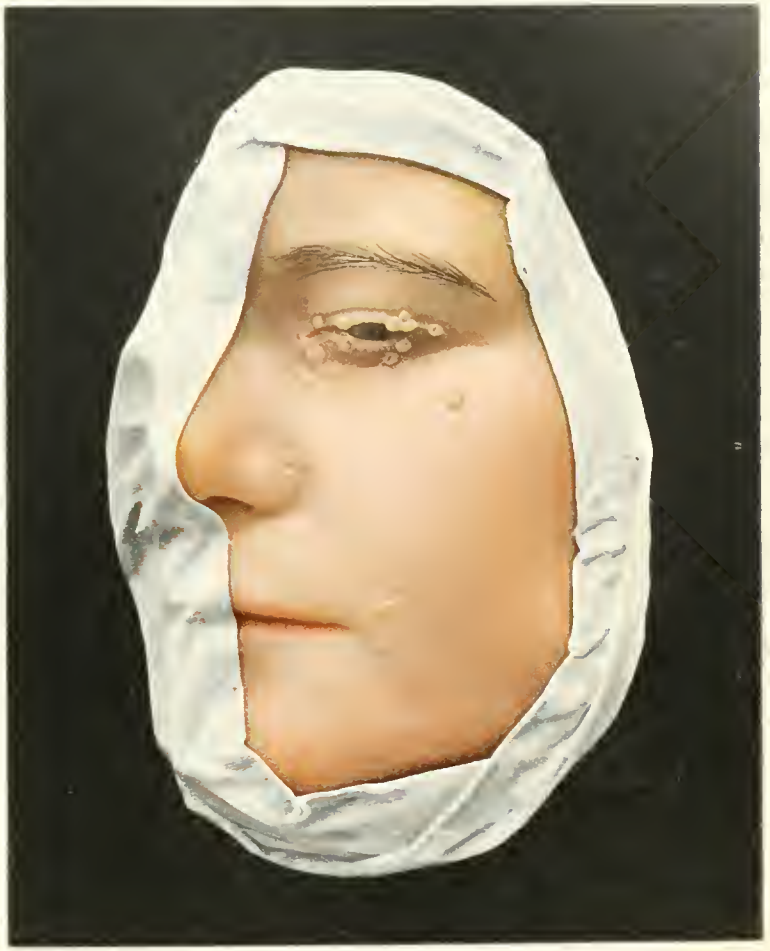

Fig. 14.
Molluscum contagiosum.

# Atheromatous Cyst of the Margin of the Eyelid.

Plate XI., Fig. 13.

By atheroma (Grützbeutel) is meant a retention-cyst, developing from sebaceous glands and hair follicles, and containing a gritty, whitish mass of cornified, degenerate epithelial cells, fat-droplets and cholesterin. The cyst walls are thin, as a rule, and composed of connective tissue.

Atheromata develop usually in middle age on the hairy scalp or the genitalia. Not infrequently they are found on the margin of the eyelid, sometimes multiple, and originating in the ciliary hair follicles (glands of Zeiss). The cysts arising from occluded sebaceous glands of the palpebral margin, the so-called glands of Moll, develop only as small translucent blebs the size of a pea or cherry.

**Diagnosis** is easily established from the location, painlessness and form and is rendered certain by the contents of the cyst. In the same location there may also be found congenital dermoid tumors lying beneath the skin of the lid, usually in the upper lid in either canthus. These push into the orbit but, as a rule, so superficially that the globe is not displaced. Beneath the skin they may be palpated as easily movable tumors the size of a bean.

**Therapy** is surgical, but the extirpation must be thorough, for otherwise they are apt to develop again.

# Molluscum Contagiosum.

Plate XI., Fig. 14.

Molluscum contagiosum (Cf. Epithelioma) is a growth composed of elevations, generally hempseed in size, rarely as large as peas, yellowish-white in color and occasionally of the hue of mother-of-pearl, centrally depressed. From this depression or crater, a gritty matter may be squeezed. These growths occur anywhere on the skin but are most common on the genitals and the eyelids, and close observation will detect them in the latter location oftener than is supposed—usually multiple on the lid margins.

The tumor is contagious and hence, in persons of uncleanly habits, apt to take on multiple form. If such a growth exist on the margin of the upper lid, it is not long before one develops on the lower lid at the point of contact. A blind individual whom I saw had many hundred mollusca.

Retzius was the first to demonstrate their contagiousness by successful inoculation.

The contents expressed from the nodule contain, histologically, cornified epithelia and numerous oval, very refractive, sharply defined bodies, the so-called molluscum corpuscles,—easily seen in sections of the extirpated tumor. Virchow, Caspary and Lasser consider them bladder-like formations due to an alteration of cell-protoplasm. According to Bollinger, they are unicellular parasites, gregarinæ, whilst Neisser considers them epithelial cells filled with gregarinæ. Croker takes them to be similar to the oviform cocci-

dium described by Leukhardt. Recently the growth has been carefully studied by Muetze under the direction of Uhthoff and Axenfeld and, according to him, all of the transitions from normal epithelia to the molluscal corpuscles are observable. The latter, then, are to be considered as degenerate epithelial cells and not as protozoic. What the contagium is, we do not know.

The **diagnosis** is easily gained from the form of the tumor with the central depression or crater, from which a core may be expressed in which, microscopically, the molluscal corpuscles are demonstrable.

**Therapy.** Expression of the contents seldom suffices. It is best to remove the nodules with scissors.

# Hordeolum.

Plate XII., Fig. 15.

Hordeolum or sty (Gerstenkorn) is the name given a small inflammatory swelling on the free outer margin of the lid, due to suppuration of sebaceous glands and is merely, in other parts of the body, the dermatologic acne vulgaris, simplex or pustulacea. The sebaceous glands of the free palpebral margin correspond to the hair follicles of the eyelashes and lead to them. Anatomically, they are here called the glands of Zeiss.

The sty, however, is differentiated from ordinary acne by the fact that, although a harmless affection, it causes many more symptoms and more inconvenience and pain than acne,—due to the anatomic structure of its site.

Usually the first symptom of the coming hordeolum is a diffuse swelling and redness of the whole lid with a sensation of tension soon becoming painful. Examination of the lid commonly discloses a small inflamed nodule on the free margin between or in front of the eyelashes, hard and extremely sensitive to the touch. Extension of the infiltration from the glands into adjacent tissues enlarges the nodule which may become the size of a pea or larger. As a rule, the palpebral skin is very red, the lid swollen and there is considerable pain so that the slight local affection may become very annoying. After a few days the centre of the infiltration develops a yellow point of suppuration which soon breaks externally on the palpebral mar-

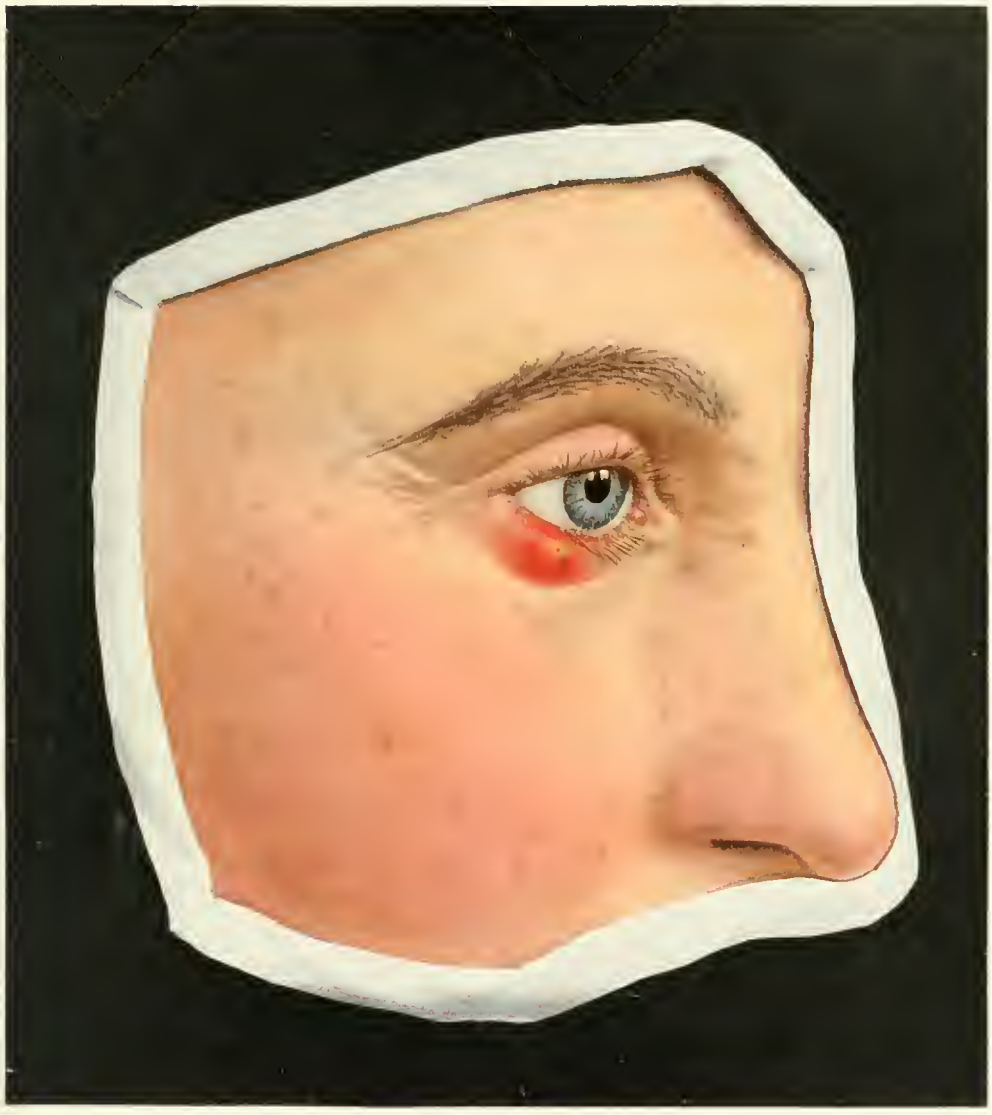

Fig. 15.
Hordeolum

gin; the pus flows out and there is healing within a few days. More rarely the infiltration extends further into the palpebral tissues and develops severe inflammation somewhat like a furuncle. In such cases the inflammatory phenomena may be quite violent and we have chemosis and marked infiltration of the palpebral and bulbar conjunctivæ as well as tumefaction of the lid.

The course is always rapid and favorable. Later, only close observation detects the slight scar left by the preceding disorder.

**Diagnosis** at first is not always easy, for the violent onset, the considerable and rapid swelling and the pains might presuppose some grave affection of the eye. A sty is to be suspected as soon as it is ascertained that the cornea is clear, the pericorneal tissue not injected, and the conjunctival sac free from abnormal secretions, thereby excluding a commencing blenorrhea as well as a deeper-seated morbidity (panophthalmia). It will not be long before a small inflamed nodule will be found near the palpebral margin, rendering the diagnosis certain. If the finger be gently passed over the surface of the lid, it often locates the swollen, painful spot.

The hordeolum generally occurs in youth, from 12 to 25 years, seldom after this period. Its genesis is favored by an often insignificant chronic blepharitis, which has given the micro-organisms (sometimes hyphomycetic) always present and abundant on the palpebral margin opportunity to multiply enormously, penetrating and occluding the excretory ducts of the hair follicles. The stasis of their secretion together with bacterial activity soon develops suppuration.

**Therapy.** When the process is incipient an attempt may be made to scatter the infiltration by warm

compresses before the glands develop suppuration. This is rarely successful, yet the warm applications are commendable, for they lessen the tension and swelling of the lids, diminish the pain, and aid and accelerate the breaking through the skin of the pus. Warm or hot cotton compresses dipped into a 2-4% boric acid solution are well borne by the eye, usually afford much relief and are much better than the ordinary applications of chamomile tea or lead-water which often contain impurities or throw down a precipitate. The compress is applied several times during the day for a quarter or half hour. If the patient remain in the house it is advisable to wear continuously a warm moist compress protected by rubber tissue, which will keep it moist 6 to 12 hours. At night this may be renewed and a bandage applied to hold it in place. If spontaneous rupture occur, the cavity should be well emptied, applying, if necessary, pressure with the finger, after which the slight wound soon heals. If the rupture delay too long and the patient desires freedom from the annoying pain, a small cut with a lancet point may be made perpendicular to the focus of infiltration on the margin of the lid and pus squeezed out, thus materially shortening the process. After evacuation the pains mostly vanish and the inflammation recedes rapidly. In such case the moist compress should be continued for another 24 hours.

The task now before us is to combat the so frequent relapses or implication of other hair follicles. It should first be determined if there be a chronic blepharitis. This, naturally, demands treatment, and the patient should also be warned to avoid for some time such external harmful influences as impure air, tobacco smoke, coal-dust, etc. Before retiring at night the eyes should be cleansed with a cloth wet with some

collyrium, *e. g.* lotio Kummerfeld, or, a thin layer of white precipitate ointment may be applied to the lids. In obstinate cases, paint the palpebral margin once daily with a 1% solution of silver nitrate.

# Chalazion.

Plate XIII., Figs. 16-17.

By chalazion or Meibomian cyst (Hagelkorn) is meant a circumscribed swelling on the inner surface of the eyelid beneath the conjunctiva. Its starting-point is a Meibomian gland lying in the tarsus under the conjunctiva and hence the chalazion develops from the tarsus. The Meibomian glands are merely modifications of the sebaceous glands of the external skin to which they are similar in histologic structure. Hence, pathologic processes in both species are much alike.

Chalazion is a chronic affection of the Meibomian glands, developing slowly with almost no inflammatory symptoms, and possibly remaining unchanged for years. During a period of months a small nodule grows in the palpebral tissue, at first causing no trouble and hence noticed by the patient only after it has attained some size. The lid is not reddened and the skin remains normal. If the tumor be minute, so that nothing is observable on the outer surface of the lid, the finger discovers the small spheric growth beneath the skin which is movable over it. Since the chalazion always develops in the tarsal cartilage, it is always mobile with the cartilage, not upon it. In the course of time the growths may become as large as peas, cherry-pits or beans. They cause then a palpebral deformity, particularly when, as is often the case, there is multiple formation, and the shot-like nodules may be seen from afar protrud-

Fig. 16. Blepharitis marginalis sicca. Chalazion

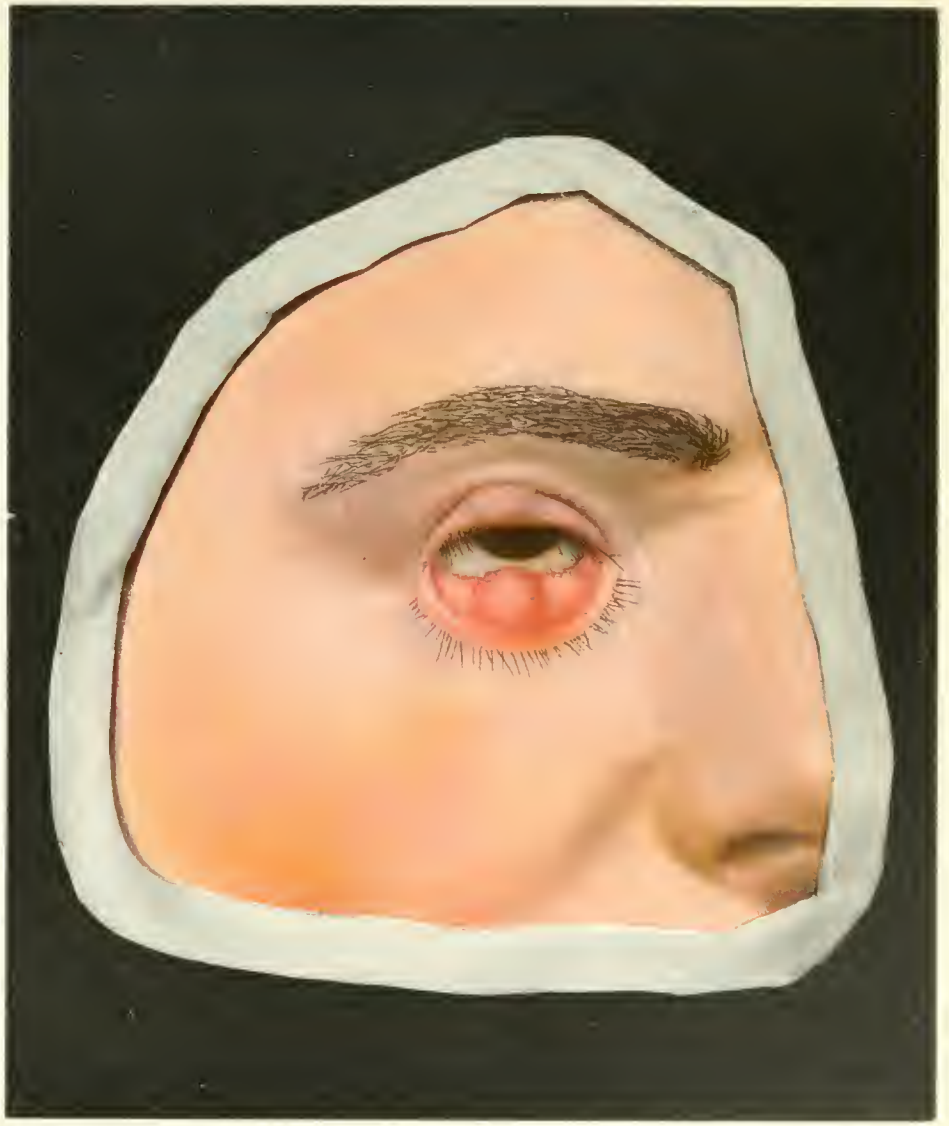

Fig. 17. Chalazion (seen from within).

ing outward beneath the skin. Even in this stadium inflammation of the lid or pain is absent. If the lid become ectropic, a condition often aggravated by the stiffness of the palpebral tissue, we note a more or less prominent yellow-brown or slate-gray fleck with a reddened areola in the conjunctiva projecting into the eye. This may finally break through the conjunctiva, after which a somewhat thick, slimy fluid exudes from time to time and the tumor decreases somewhat in size. Its major portion, however, composed of firmer granulation tissue, remains in its capsule unchanged. In the course of months or years even these masses may so shrink or resorb that the tumor disappears.

The chalazion is usually noted in adults, seldom developing in children. Generally, there are several on one lid, or, all the lids are deformed by these lumps. In the beginning they cause little trouble, but in the advanced stage are disfiguring and either by the development of inflammatory symptoms, or by mechanical hindrance to the movement of lid and eye greatly annoy their possessor.

Chalazia develop mostly where there exists a slight but chronic conjunctivitis, leading to occlusion of the excretory ducts of the Meibomian glands and retention of their secretions. The content of the gland then becomes thickened and harder and may change by deposition of calcareous salts into a hard, chalky mass (calcareous infarct of the Meibomian gland or lithiasis palpebralis). These are seen beneath the palpebral conjunctiva as small white or bright yellow spots. In fact, calcareous infarcts of the Meibomian glands are frequently prodromal of chalazia, and are usually noted in considerable number surrounding a chalazion beneath the conjunctiva of the same lid. These thickened masses of glandular secretion may exert an inflammatory action upon the endothelium and adjacent

tissues, because of which these begin to proliferate and become infiltrated with small cells. With the progress of such infiltration, the mass develops into a dense granulation tissue in which, sometimes, even giant cells are found. The center of this granulation tumor, deprived of vascular supply, may finally disintegrate and pass into mucoid degeneration. A dense, tough capsule, formed of the surrounding tarsal tissue under pressure, commonly develops about such a mass, and the chalazion, therefore, is composed of tough, dense granulation tissue enclosed in a fibrous connective tissue capsule.

Recently it has been maintained by several authors that the chalazion is, as a rule, tuberculous in nature, a statement which has been further supported by the demonstration of numerous giant cells in its tissues. Many investigations, however, prove the fallacy of such assertion, at least in the majority of cases, and the benign clinical course scarcely bespeaks a tuberculous process.

**Therapy.** At the beginning and as long as the chalazia remain small, we may try to scatter them by external means, *e. g.*, by rubbing an ung. potass. iod. into the conjunctival sac or by painting the external skin with tr. iod. If this have no effect, small, hard chalazia may be left undisturbed. If they grow or are already so large as to be disfigurative, operation is indicated.

The query at once arises whether the incision shall be dermal or conjunctival. Although the protrusion is mostly outwards, the skin should never be incised. The natural opening is inward, toward the conjunctiva, and by this path the tumor-masses are most easily reached and there results minimal deformity from the operation. The operation is not so easy and simple as

it might seem, for a single incision does not suffice and the removal of the tough tissue is essential and often difficult. The first cut, therefore, should by no means be too small or superficial. Its direction should be parallel to the palpebral margin.

As the operation is painful, we anesthetize by placing in the conjunctival sac three or four times at minute intervals some drops of a 2-4% solution of cocain, or, better yet, by injecting with a Pravaz syringe a few drops subcutaneously at the site of the tumor. The ectropic lid is best fixed with a blepharostat, the conjunctiva is divided with the knife down to the capsule and we endeavor to dissect out with forcep and scissors the encapsulated node. This is not always easy for the capsule is apt to be firmly attached to the surrounding tissues. If it cannot be shelled out in its entirety, as much as possible is cut out with scissors and forcep and the remainder removed with the sharp curette.

To avoid relapses and the formation of new chalazia, a chronic conjunctivitis present must be treated and cured. If white calcareous infarcts of the Meibomian glands are noted, so that a number of excretory ducts are occluded, the glands must be slit open by passing a cataract needle down through the conjunctiva until the calcareous mass is exposed, when the infarct may be removed with a fine curette.

# Blepharitis Marginalis.

Plate XIII., Fig. 16; Plate III., Fig. 3; Plate XIV., Fig. 18; Plate XXVIII., Fig. 41; Plate XXIX., Fig. 42.

The various affections of the palpebral margin which begin with symptoms of an inflammatory nature, we term blepharitis marginalis. They belong to the most common diseases of the eye seen by the general practician, particularly in the larger cities and among the poorer classes where the anemic and scrofulous children compose a majority of such patients. The skin of the body becomes very thin and delicate on the eyelids and still more so as it approaches the margins so that here we have the most tenuous and sensitive derm of the whole body. For this reason, it is easily comprehended that in the most various dermal diseases, particularly if located on the face and extending therefrom, we often find the palpebral margin particularly inclined to sympathetic complication. The various skin troubles appearing on the eyelid and its margin are not, as a rule, differentiable from the adjacent foci of disease and should receive similar treatment, in regard to which special works are to be consulted.

There frequently appear, however, on the margin of the lid characteristic types of inflammation which here demand special consideration.

Firstly, we should differentiate from true inflammations of the palpebral margin, a hyperemia of the part, well-termed blepharitis vasomotoria.

*Hyperemia marginalis.* In many delicate-skinned individuals, and particularly in the blond, the marginal skin is so sensitive that it becomes very red from the least external stimulus or irritation. As soon as these patients enter an atmosphere of tobacco smoke or go out in windy weather or are exposed to a strong light, they develop the ugly "red eyelids" within a few hours or the next morning. The phenomenon not only disfigures, thus often spoiling the patient's enjoyment of some harmless pleasure, but is also accompanied by many inconveniences. The eyes itch and burn, forcing the patient to rub the margins of the lids, and furthermore there is a sensation of weight and heat in the eyes often extremely annoying when engaged in difficult work. Often it requires no external irritant to evoke the troublesome symptoms which may be caused by unusual bodily exertion, overuse of the eyes, emotional disturbances, etc. If we consider that in most individuals, excesses, a night's carouse, long exposure to impure air laden with tobacco fumes are quite evident in the eyes the morning after, it is comprehensible how vexatious it is for most youthful patients with hypersensitive margins of the eyelids to go about after the least indiscretion with swimming, reddened eyes which seem to betray an over-indulgence in alcohol or a night spent in tears. Not infrequently the insignificant affection hinders them in business.

In acute attacks, the margins of both lids are much reddened and if closely observed, there will be found in the redness a number of delicate, bright-red, deeply-injected blood-vessels. Coexistent there is often present a slight swelling of the lids and an injected palpebral conjunctiva. Scale formation on the margin of the lid or at the roots of the lashes is usually lacking, but the lacrimal secretion is, as a rule, increased, so that the eye "swims in tears."

If the trouble has been of some duration, the acute attacks, at first often repeated, develop a chronic condition, *i. e.* the lids remain red and their margins become thickened and heavy. Many thick, distended blood-vessels are seen in the palpebral edge which passes from a red to a violet tint. The patient develops great photophobia, and, because of the ocular trouble, have to be extremely careful of themselves. Even at some distance the ugly red margins are visible.

The affection, at first insignificant, is most persistent, and in many instances resists treatment for a long while. Therapy must be both general and local. It is most important to strengthen and harden the young, delicate, often anemic or scrofulous individuals in whom the trouble first begins. Although it is necessary to warn against excesses, over-exertion, late retiring, too long reading, exposure to impure air, it is equally essential to avoid coddling. On the contrary, these patients should be as much as possible in the fresh, open air, and, healthful exercise, cold affusions with vigorous massage, bathing out of doors will in time strengthen the organism and harden the sensitive skin. Tonics such as quinine, iron, etc., may be given internally.

Locally, the parts should be kept clean, and cold compresses of some mild astringent, such as lead-water, very weak solution of tannic acid, water containing a few drops of eau de Cologne or ethyl alcohol are to be commended. Whatever the agent, care should be taken that the sensitive parts be not irritated too much, and strongly concentrated solutions are to be avoided. The eye-douche is very useful and should be employed once daily or every second day, a finely divided and not too forcible stream being directed for 3-6 minutes against the edges of the gently closed lids.

To the douche may be added any suitable astringent and hardening agent (eau de Cologne, alcohol, borax).

Salves are best dispensed with, for the ordinary unguents are much too irritant. If the skin of the lids shows a tendency to chap or crack, a very thin film of pure lanolin may be applied at night before retiring.

In obstinate cases, the margin may be painted with a 1% solution of silver nitrate or 2-3 superficial application made of lapis mitigatus in substance.

Of inflammations of the palpebral margin, we have two chief types for differentiation:

1. *Blepharitis marginalis sicca*, also called blepharadenitis, seborrhea marginalis, blepharitis squamosa, is a condition of hypersecretion from inflammatory irritation of the sebaceous glands of the palpebral margin, and hence, more exactly, a seborrhea of the ciliary portion of the eyelid. The sebum soon dries and forms small scales lying between the eyelashes upon the skin of the lid. Recent investigations have demonstrated that these scales are not altogether the product of desiccatetd sebum and dead, cast-off epidermal squams, but that numerous hyphomycetes and their colonies found on the margin of the lid and in the excretory ducts of the glands probably engender the disease in most instances.

The patient is usually driven to the physician because of a continual itching and burning. If the margin of the lid be superficially examined, little that is abnormal is observed, but on closer investigation, or if one rub the finger firmly across the eyelashes, the numberless minute, whitish-gray scales lying upon the margin of the lid at the roots of the lashes will come into evidence. After such dry massage, the palpebral margin looks as if powdered with flour, and by such pulling and rubbing the lashes may be dusted off. Underneath the scales the margin of the lid is reddened

but not ulcerated. The cilia are loosely rooted and easily removed, but in recent cases grow in again as before. When the condition has been of longer duration, the eyelashes are affected, lose their luster, become bent and twisted and finally fall out. Formation of crusts or scabs with a glueing together of the lashes seldom occurs. In such case, the crusts are chiefly composed of the dried mucous secretions of the glands, and beneath them (in blepharitis sicca) there is no ulceration.

In **therapy** it is to be well understood that no unguent nor any other remedy is of the slightest value unless before each application all scales are removed and the lid margin most carefully cleansed. The scales are best disposed of by dropping a little pure olive oil upon the margin and rubbing it in between the lashes. After a few minutes the scales have become loosened and may be removed by rubbing with a piece of flannel and using ciliary forceps until the field is clear. Repetition of this process as soon as new scales are formed should not be neglected because of the slight, transitory swelling and redness of the lid margin following its execution, nor is any harm done if some lashes fall out; when loosely rooted they come out sooner or later, growing again as soon as the margin of the lid becomes healthy. After cleansing and drying the parts, a portion of salve the size of a pea is rubbed into the margin with a glass rod or the finger. Since cleansing, in the first sittings, somewhat irritates the eye, it is best done once a day, before retiring. The salve remains upon the margin of the lid in a thin layer during the night, and is washed off in the morning with soap and water. Suitable ointments are a 1 to 2% ung. Pagenstecheri (hydrarg. oxyd. rubr.) or ung. diach. Hebrae, best attenuated with equal

quantities of vaseline. Carefully treated, blepharitis sicca is not obstinate and soon heals, without sequelæ.

2. *Blepharitis eczematosa* or blepharitis ulcerosa, scrophulosa, is, as its name indicates, an eczema of the palpebral margin, and, as in dermal eczema, exhibits the most varied types.

The first three stadia develop rapidly, as a rule, or else are not distinctly observed as such; the fifth stadium begins in an eczema already in the process of healing, so that the physician is best acquainted with the fourth stadium, by far the commonest in most cases, the stadium of moist ulceration and crust-formation.

Primarily we have a hyperemia and swelling of the lid margin, due to a serious saturation of tissues and leucocytic emigration. The tumefaction usually develops in spots so that a number of small, dense red nodules, the size of a hempseed or pinhead are found on the margin (stadium papulosum).

With an increasing serous infiltration, the epithelium of the derm is raised up here and there and circumscribed collections of fluid form between the epithelium and the rete mucosum, vesicles filled with a clear watery fluid (stadium vesiculosum).

Gradually the leucocytic emigration augments until the contents of the vesicle become more and more turbid and finally purulent (stadium pustulosum).

At last the pustules break and ulcers form, soon crusting over. Beneath the crusts the weeping ulcers persist unchanged (stadium madidans), and in this state the disease may continue for a long time, new vesicles and pustules forming in the neighborhood of the ulceration, so that the various stadia may be synchronously observed, the stadium madidans predominating.

When finally the inflammatory phenomena subside,

the exudation and crust-formation lessens. The ulcers heal, and epithelial loss is no longer observed on the superficies of the derm, but, the skin retains for some time the inclination to develop inordinate quantities of epithelial cells which rapidly cornify and are cast off. Hence, we find the affected areas covered with layers of scales, the stadium squamosum.

As mentioned above, the physician is usually confronted with the matured eczema in the fourth stadium. The margin of the lid is markedly swollen, thickened, and covered with crusts, and not infrequently vesicles and pustules are seen in the neighborhood of the crusts. The pustules are most commonly located about the cilia, and when they rupture the single cilium is observed rising up out of a deep, crater-like ulcer. The ulcers soon begin to exude, and thick crusts form, which, if removed, expose the deep, easily bleeding ulcus. If the disease remain untreated the lashes fall out and do not return. The angle of the lid-margin is eaten away so that a slight eversion results. The loss of the cilia is due to the fact that their follicles have been destroyed by suppuration. Where this has not happened, the cilia grow again through the crusts and cicatrices, but pervertedly, so that the eye may suffer greatly from their abnormal positions (*vide* Plate XXII., Fig. 13). Almost invariably the remainder of the palpebral skin and the conjunctivæ are affected, and commonly the eczema extends to the cheeks or nose. Eczema of the scalp as well as of the eyelids is often present. The nasal cavities should be watched most carefully, for in them analogous processes (eczema, purulent discharges, ozena) often develop.

As the cause of the disease, we almost invariably find a general scrofulosis and tuberculosis. The individuals affected are usually frail, poorly nourished

children, exhibiting all the signs of scrofula (eczema, glandular swelling, a puffy, bloated appearance, thick lips, etc.) More rarely, local injurious influences (bad air, dust, occupation-noxæ) or persistent conjunctival irritation from epiphora or other secretions may develop eczema and ulceration of the palpebral margins.

The **diagnosis** of blepharitis eczematosa is not difficult. The disease is differentiated from blepharitis sicca, which occasionally begins with crust-formation, chiefly by the fact that when the crusts are removed, the deep eczematous ulcers appear. The eczema might be confused with sycosis (Bartflechte) which sometimes locates on the margin of the eyelid, but in sycosis the large, exuding areas and ulcerations are absent. Furthermore, sycosis develops almost invariably in adult males, whilst blepharitis eczematosa usually affects weakly children.

The *course* of an untreated blepharitis eczematosa is extremely chronic. In its chronicity it passes into a stage where the epithelial layer of the skin becomes necrotic, the denuded areas covered with thick, solid, yellow-brown scabs, and in this state, the disease may persist for years.

With long continuance of the affection, the eyes suffer in many ways, and there are a number of sequelæ of chronic blepharitis which injure the visual organs to a greater or less degree, *e. g.*

Chronic conjunctivitis, which may cause much trouble.

Destruction of the cilia and the margin of the lid. The hair follicles and glands become implicated in the ulcerative process and are destroyed by suppuration. Finally, all of the lashes fall out or there remain only a few. The lid-margin breaks down, and instead of the normal rectangular form, its delicate edge be-

comes rounded off, so that some portions are shrunken, others hypertrophic and thick.

Trichiasis. The few remaining cilia may be turned inward by cicatricial contraction and thus possibly abrade the cornea.

**Therapy** should not only be local, but constitutional. The hygienic environments of the patient are to be improved, the delicate children properly nourished, mountain or seashore prescribed when conditions permit, and every endeavor made to vanquish the existent scrofulosis. Internally, cod-liver oil, iron, iodine, etc., are indicated, and for the habitual obstipation often present, calomel should initiate the treatment. Above all, the rarely absent nasal complication must be cured, for as long as it is present an apparently healed eye will soon become diseased again.

Of local measures, the first is a careful, daily removal of the crusts. This is accomplished by loosening them with warm water, or still better, with olive oil, followed by rubbing or scratching them off with blunt forceps. Loose or slanting lashes should be extracted with cilia forceps. One should not be frightened if the ulcers beneath the crusts bleed easily and reproduce the crusts. In the first days of treatment a light superficial brushing-over of the ulcer's base with the mitigated silver nitrate stick (lapis mitigatus) is often helpful. Unguental treatment is particularly applicable in this disease, and, as in all eczemas, care must be taken not to use too irritant salves. The choice of the salve is not of so much consequence as its degree of concentration. In weeping eczema of the eyelid and margin, it is best to prepare a compress suggested by Hebra. The ointment is spread thick upon strips of boracic gauze and these applied shingle-fashion to the eye, *i. e.* one strip overlying the strip

below. In order not to annoy the patient too much, the eyes may be treated in alternation. The best unguent is Hebra's diachylon salve; the white and red precipitate ointments are often used but it is wise to prescribe the latter in less strength than originally given by Pagenstecher. Schreiber, of Magdeburg, is very successful with a ¼% ointment of silver nitrate. A 1% resorcin unguent is recommended where the eyes are hypersensitive. The bandage should first be changed once, later twice in the 24 hours, and it is important to see that the unguent be freshly made and its base not rancid.

This unguental treatment generally causes the eczema to pass from the stadium madidans into the stadium squamosum or desquamation, when it should be stopped, the affected areas of skin usually healing soon with the employment of a dusting powder.

In particularly obstinate or chronic eczemas, a tar treatment after the unguental is often advisable. The affected areas are painted daily with pure tar or with equal parts of tar and olive oil.

With needful patience on the part of patient and physician and some care in the use of salves, which are not equally well borne by all patients, it is possible to overcome the most obstinate forms of the disease.

# Entropium.

Plate XIV., Fig. 18; Plate XXII., Fig. 31.

In entropium, the lid or its margin is turned in so that the free edge and the lashes no longer project outwards but lie directly against the eyeball, upon which the cilia rub with every movement of the lid, thus causing irritant and inflammatory phenomena, *i. e.* they act like foreign bodies upon the exterior envelope of the globe.

Palpebral entropium is differentiated from trichiasis and distichiasis as follows: In trichiasis the lashes are correctly placed but the margin of the lid bearing them rolls inward toward the globe whilst in distichiasis, the margin lies normally but the cilia grow crookedly or too much in an inward direction so that they impinge upon the eyeball. Frequently, however, we find both conditions present: misplacements of margin and cilia.

If in a case of marked entropium we look directly at the eye, the edge of the lid is scarcely visible and it is only when the lid is pulled outwards by the fingers that it unrolls, bringing the margin into view, and, when let go, it rolls in again and the margin vanishes.

The entropium may involve the entire lid, or portions only may be turned inwards, a partial entropium. In case of the latter, the lower lid is chiefly affected in the middle and outer third; the upper lid mostly in the outer third. Both lids, the upper and the lower, are about equally subject to the disease.

Various degrees of inversion may be differentiated.

Fig. 18.
Entropium Trichiasis following
Conjunctivitis simplex chronica.

In the minimal degree the free edge of the lid is turned so far inward that the tips of the cilia impinge upon the eyeball, being so twisted that they lie nearly parallel to the lid margin and glide tangentially across the globe. If the lid margin is turned so far inward that the lashes rest directly upon the eyeball, they gradually assume a position the reverse of natural, *i. e.* they no longer curl outward but are bent inward to correspond to the curve of the eyeball so that their tips are directed away from the cornea toward the conjunctival fornix. In the maximal degree of inversion, the palpebral skin lies against the globe whilst the cilia penetrate deeply into the conjunctival fornix.

Entropium always results in injury to the affected eye, chiefly caused by the internally directed cilia. The patient has the sensation of a foreign body in the eye, and soon there develop extreme lacrimation, photophobia, conjunctival and pericorneal injection. If the entropium persist for some time, the cornea, in particulate, suffers, and may be injured permanently. Because of the continual irritation, the corneal epithelia become indurated, thickened and cloudy, or, it may happen that the epithelia in divers areas are injured by the cilia, and if opportunely infected, a superficial ulcer of the cornea may develop. In old chronic cases, the cornea is generally covered with pannus-like opacities and indurations.

Inversion of the lids is invariably the result of previous definite diseases of the eye, and in most instances is due to contraction or shriveling of conjunctival scar-tissue. According to their etiology we differentiate two chief forms of entropium:

1. *Entropium cicatriceum*, where, by cicatricial contraction of conjunctival tissues, the margin of the lid is drawn inwards, the scars being due, generally, to a long-lasting trachoma, to the so-called cicatricial tra-

choma (*vide* Plate XX., Fig. 31). Each trachoma granule heals cicatricially, whence, where many and recurrent granules have occupied the palpebral conjunctiva, the conjunctival tissue still left, does not suffice, because of the long, radiating scars extending chiefly in a horizontal direction, to cover without tension, the entire inner surface of the lid. Consequently, the lid is constricted interiorly from above downwards, and the punctum mobile, the free margin of the lid, is forced to yield and roll inwards. In such cases, the cilia, impinging almost entirely upon the eyeball, are no longer normal, but develop as long, fine hairs or degenerate into short, thick stumps (trichiasis). The free angular margin of the lid is, generally, no longer present, having been rounded off or quite flattened out.

In other cases the conjunctival scars causing the entropium were due to wounds or burns or cauterizations.

2. *Entropium spasticum.* This follows spasmodic contraction of certain fibers in the musculus orbicularis. To elucidate the genesis of this form of inversion, we must consider briefly the structure and action of this muscle. The musculus orbicularis is a superficially extended muscle of the skin, divisible into two portions. The first or inner portion, lying within the lid itself, extends to the palpebral fissure; which it encircles, and is, therefore, called the portio palpebralis. The second, or outer portion, extends peripherally from the first to the orbital edge and adjacent parts and, hence, is dubbed the portio orbitalis. As a rule only the palpebral portion governs the movements of the lid; the orbital portion merely draws together the facial derm surrounding the eyes and, therefore, aids in firm closure of the lids. The fibres of the portio palpebralis have a double curve: 1. with a concave side toward the palpebral margin, and 2.

with a concave side toward the eyeball, corresponding to its curvature. A contraction in these muscle bundles would result in straightening out both curves (toward the concave side), thus first closing the lids and, secondly, pressing them against the eyeball. If now, from any cause, there develops an unequal or imbalanced contraction in the fibers of the orbicularis, *e. g.* so that the fibers at the lid margin are contracted or spasmodically tense, whilst all other fibers remain lax, the palpebral fibers overcome the others and roll the margin inwards. To accomplish this, however, another factor is requisite, namely, that the palpebral derm be not tense but loose and flabby. Both of these conditions are often present in elderly individuals, for which reason, entropium spasticum is most commonly found in this class of patients (entropium spasticum senile), particularly where the eye has been kept closed for some time, as after cataract operations, when it oftener occurs and becomes a very troublesome complication.

In like manner, the orbicularis fibers of the margin of the lid gain the ascendancy if their tension be not counterbalanced by a normal curvature of the eyeball, which, if lacking, almost invariably develops inversion of both lids. But, even after the retrogression of the bulbus into the orbit, often occurring in emaciated seniles, entropium may develop, and likewise after a shriveling and atrophy of the bulbus.

From a spasmodic contraction of the orbicularis (blepharospasmus) found particularly in children, entropium may be engendered, and here the portio orbitalis often acts so violently that the margins of the two lids are pressed against each other until finally one of them rolls inwards. This form of entropium spasticum in youthful patients affects the lower lid alone.

**Therapy.** Entropium in old people due to the

pressure of a bandage commonly disappears when the bandage is removed, but, if for other reasons it is necessary to continue the compress, we may endeavor to hold the lid in its normal position by using adhesive plaster and collodium, the application beginning at the palpebral edge and extending downward over the cheek. If the lid be not kept in place by this method, ligation will be necessary.

If the inversion be due to blepharospasmus, this should be treated and cured, after which the entropium disappears of itself.

In entropium due to cicatricial contractures, it is first essential to remove carefully with cilia forceps all the eyelashes impinging upon the bulbus. A permanent cure is attained only by operation.

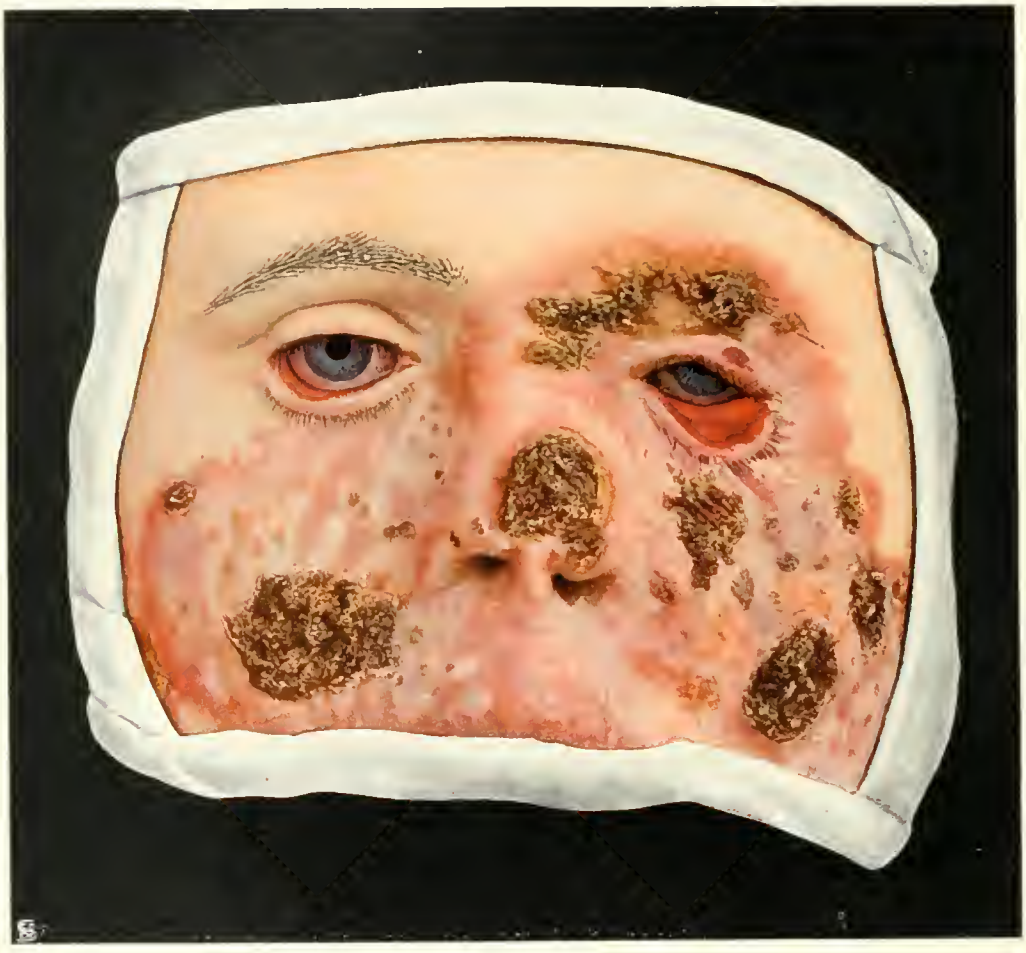

Fig. 19.
Lupus vulgaris faciei. Narbenectropium.

# Ectropium.

Plate XV., Fig. 19.

By ectropium is meant an anomalous position of the lids where they with their conjunctivæ are no longer closely applied to the bulbus but roll outwards, away from it. When this happens, a larger or smaller area of the conjunctiva may lie exposed to view. There are various degrees of eversion, from the slightest, where the palpebral margin does not quite touch the eyeball, to a degree where the whole lid is turned inside out. One or both lids may be affected; most commonly, however, only the lower.

But, even with a minimal eversion, the condition is extremely troublesome, and commonly the ectropium with its sequelæ soon develop a state of great irritation in the eyeball. Then, with the separation of the lid margin from the globe, the lacrimal puncta are thrown outwards and no longer drain the lacus lacrimalis. The natural transit of the lacrimal fluid being thus interfered with, the tears trickle over the edge of the lid and down the cheeks, and we have epiphora.

A further consequence of ectropium is the inflammation and hypertrophy of the conjunctiva thus exposed to external irritants—air, dust, etc. It tumefies, and the swelling leads to yet greater eversion. Thus, both conditions alternately aggravate each other. A high degree of ectropium may finally develop grave consequences for the eye, since the cornea, not being adequately covered by closure of the lids, invites the development of a keratitis e lagophthalmo.

According to their etiology we differentiate several species of ectropium:

1. *Ectropium paralyticum.* After paralyses of the musculus orbicularis, *e. g.* as a complication of facial paralysis, there usually develops a slight eversion of the lower lid, explained as follows: The orbicularis whose fibers are paralyzed is no longer able to hold the lid accurately against the globe; hence, the lid, obeying the law of gravitation, sinks down and somewhat away from the eyeball. Because of this mechanical genesis, it is clear that the lower lid alone will be affected.

2. *Ectropium senile.* This form is very similar to the one just described, both in genesis and appearance, except that here paralysis is not etiologic but rather the lax, senile skin and musculature of the lid which are no longer able to hold it firmly against the globe. The lid sinks somewhat down and outwards, and there develops a sulcus betwixt lid and globe in which the exposed conjunctiva may be seen.

3. *Ectropium spasticum.* Found chiefly in children and youthful individuals suffering from an acute conjunctivitis with tumefaction, particularly when accompanied by a blepharospasm. If, with such children we attempt a forcible opening of the eye, it happens that by a strong contraction of the orbital portion of the musculus orbicularis a sudden and spontaneous eversion of both lids occurs. This may also be excited by simple pressure on the lids without touching the eye. For the development of an ectropium spasticum a conjunctiva well tumefied and a spasmodic state of the musculus orbicularis, more marked in the portio orbitalis, are necessary. If such a suddenly developed eversion be not speedily corrected, the already swollen conjunctiva will be strangulated by the portio palpebralis of the orbicularis and marked edematous tume-

faction will ensue. This may lead to permanent eversion until the conjunctivitis has been cured. Ectropium spasticum is mostly found in children suffering from a scrofulous conjunctivitis, more rarely in cases of ophthalmia neonatorum. It usually affects both lids simultaneously, and may attain a high degree, so that the bulbus is completely hidden by the swollen, everted lids.

Lesser degrees of ectropium may develop from simple swelling of the conjunctiva, which becomes not only thicker but broader and, because of the sausage-like tumefaction of the palpebral edge, is pushed still further away from the globe (ectropium mechanicum). The eversion is accentuated when, from pinching or a spasmodic statte, there is contraction of the palpebral portion of the orbicular muscle.

4. *Ectropium cicatriceum.* The highest degree of eversion is caused by cicatricial contractures. This demands that a portion of the palpebral edge have been destroyed and replaced by scar tissue. Such conditions are peculiarly apt to develop after burns of the skin of the lid or wounds of the lid, operations upon the cheek, caries of the orbital edge, etc. The scar formed invariably draws the margin of the lid yet farther downwards until finally the entire reddened and thickened conjunctiva of the lid is fully exposed outwards, and of the lid itself only the edge, now far removed from the globe, is visible.

**Therapy.** The earliest possible correction of the eversion is to be sought, for the portion of conjunctival tissue turned outwards is continually exposed to the air and the dangerous substances contained in air, and which excite violent irritation of the eye. With increase of the conjunctival swelling, the eversion augments until finally a circulus vitiosus is developed.

A treatment without surgical intervention is pos-

sible only in ectropium spasticum. Here, in the beginning of the trouble, the everted lids may be replaced without much difficulty and, to avoid an ever-ready relapse, they should be kept in the correct position by strips of adhesive plaster or the use of a compression bandage.

In obstinate cases, an extension cut of the external canthus is to be commended before the correction of the eversion, for the bleeding caused by the cut is very beneficial. After the lids are replaced, the inflammation and swelling of the conjunctiva should be reduced.

Other forms of eversion are corrected only by means of the ectropium operation.

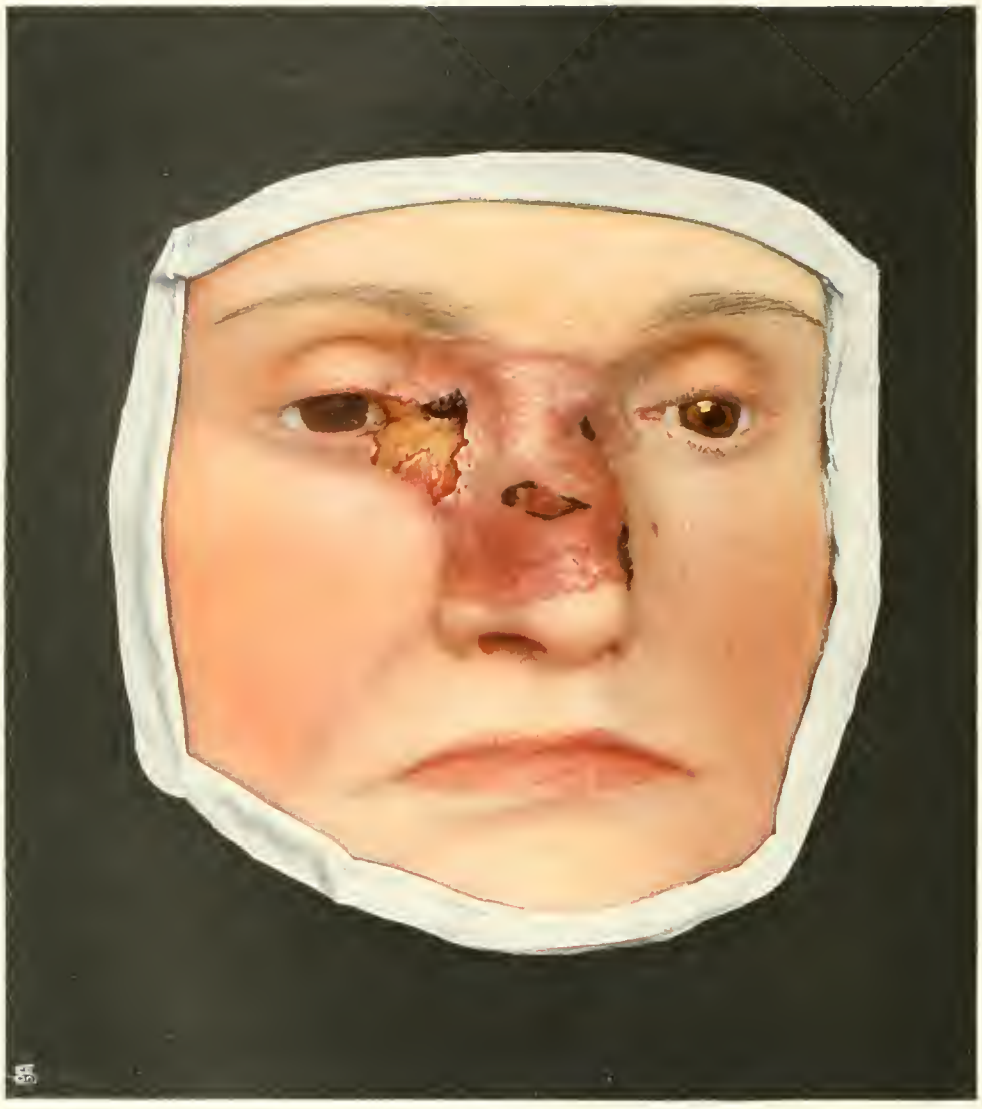

Fig. 20.
Carcinoma epitheliale I.

# Carcinoma Epitheliale Palpebrarum.

PLATE XVI., FIG. 20; PLATE XVII., FIG. 21.

Carcinoma is the commonest palpebral neoplasm. It usually occurs as a shallow ulcer, scarcely rising above the superficies and having but a slightly elevated wall-like periphery. Actual tumors are few in number. The flat, slowly extending new growth is often called "ulcus rodens," but anatomically it is a genuine cancer of the superficies.

There are two points of election for the development of palpebral carcinomata, first the inner canthus where we have a junction of external skin and conjunctiva. It is well known that such dermo-mucosal blendings (the margin of the lips, anus, palpebral margin) are areas of predilection for carcinomatous genesis. The second point of election is the external derm of the eyelid. Carcinomata developing in the first-mentioned area are usually more malignant and more rapidly and deeply destructive, whilst those developing in the palpebral derm are mostly benign and therefore of the ulcus rodens type.

As regards localization the left side of the face is more often affected than the right. It is also certain that the neoplasm is much commoner on the lower lid than the upper, and more apt to be found in the internal canthus and, as first remarked by Valude, in the neighborhood of the lacrimal sac. Here the cancerous process penetrates deeply and rapidly, destroying the lacrimal sac, then eroding the lacrimal bone

and extending to the nasal cavity, or, the tumor mass pushes through the lacrimal duct into the nasal fossa.

Fig. 20 on Plate XVI. shows one of these flat dermal cancers of the left upper lid about to pass over the bridge of the nose to the other side.

The growth of a superficial carcinoma on the eyelid may be extremely slow, sometimes covering a period of 20 or 30 years (Schulz-Zehden).

Such was the case of an old woman in the Home for Incurables, of whom Dr. Schulz-Zehden (Berlin) kindly permitted a wax model to be made, shown in Plate XVII., Fig. 21. The cancer, extending from the margin of the lid, had existed for many years. It had excavated a deep hole in the left side of the face, and the eyelids, the skin of forehead and cheek and the bony margin of the orbits had entirely disappeared. The deep-sunken, shriveled eyeball, in which the cornea is distinctly seen, still remains. The patient had invariably refused operation, keeping the eroded area covered with a moist cloth. Recently, death occurred, and when we consider the many years of its existence, the slow progress of the carcinomatous growth is truly remarkable.

In other cases the nose and cheek are soon implicated and upon them the growth progresses by continuity. Occasionally, however, autopsy alone demonstrates how extensively the adjacent organs and cavities were involved.

Often the lymph glands remain unaffected for a long period. Thiersch observed glandular involvement in two instances only; Winiwarter, in 26 cases, twice; Mayeda in his series of 195 carcinomata of this type, found eight glandular involvements, a percentage of 4.1.

In general, it may be said that the superficial type does not develop carcinomatous processes in the glands nor metastasis to internal organs.

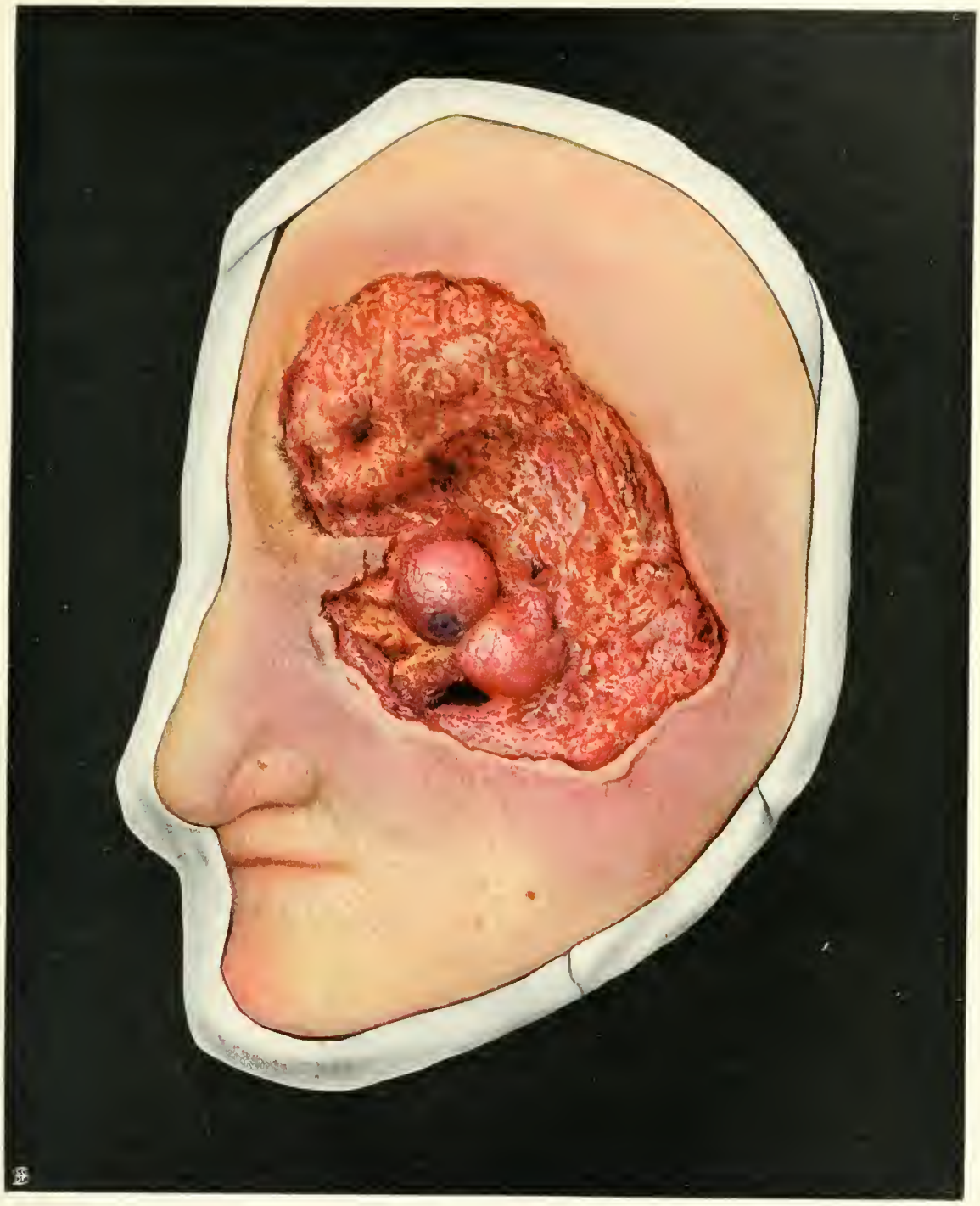

Fig. 21.
Carcinoma epitheliale palpebrarum II.

Etiologically, we need only remark that cancer in this region frequently arises from constant irritation or rubbing of warts; also from small wounds, cauterization, removal of vesicles, nodules, etc., and possibly from lupus.

The beginning of the trouble is well described by Unna: "Ulcus rodens commences as a rose-red or pearl-gray nodule the size of a mustard seed, rising a millimeter or less above the surrounding surface, with very slow peripheral extension and the development of a central depression. Thus, there are found areas, apparently but not actually cicatrized, of a gray-yellow or gray-red color, the size of a pea or covering an area equal to that of a dime or quarter-dollar, sometimes oval in contour, flat and lying in the plane of the healthy skin or slightly depressed, and limited by a delicate, ridge-like margin of the color of mother-of-pearl, from which frequently arise minute nodular thickenings. Any general thickening of the derm or any peripheral inflammation is not cognizable. Even in this stadium of nodular development, slight traumata of various parts of the nodule, usually the central portion, result in desquamation of the stratum corneum, and a dark crust or scab, formed of bloody serum and a new stratum corneum, develops. Left to itself, this crust falls off, after which the affection has its original cicatrized appearance. Repeated desquamation of the stratum corneum finally leads to permanent ulceration, and thus the second or ulcerative stadium begins."

**Therapy** is operative, followed eventually by a plastic operation upon the lid.

Recently, cures with the Finsen light have been obtained.

For amelioration of pain, the ulcerated areas are covered with bandages spread with unguent.

# Dacryocystitis.

Plate XVIII., Figs. 22 and 23.

Inflammation of the lacrimal sac seldom follows a lesion or infection of the conjunctiva but is often due to some ascending nasal affection, to stasis and decomposition of the lacrimal fluid above a stricture or to lesion of the adjacent bony structures.

Most commonly as a result of stricture below, the mucosa of the lacrimal sac will produce abundant pus which flows backwards into the eye, thus presenting a well known phenomenon which may long continue without externally visible inflammatory symptoms of importance.

The best name for the malady is, therefore, dacryocystitis chronica and not dacryocystoblennorrhea. If only the unfortunate term, blennorrhea of the eye—applied to the most heterogenous affections—could be dropped! Nowadays, we are accustomed to differentiate etiologically, and this should be done whenever possible. It is anything but commendable to lump together trachoma, gonococcal infection, pneumococcal infection, and pus in the lacrimal sac under the rubric of blennorrhea.

The chronic lesion of the lacrimal sac often does not betray itself externally save that pus is frequently seen in the eye. If, however, pressure is made upon the lacrimal sac in the inner canthus where it is crossed by the ligamentum canthi internum, it will be seen suddenly, that quantities of thick pus are exuding from the puncta lacrimalia.

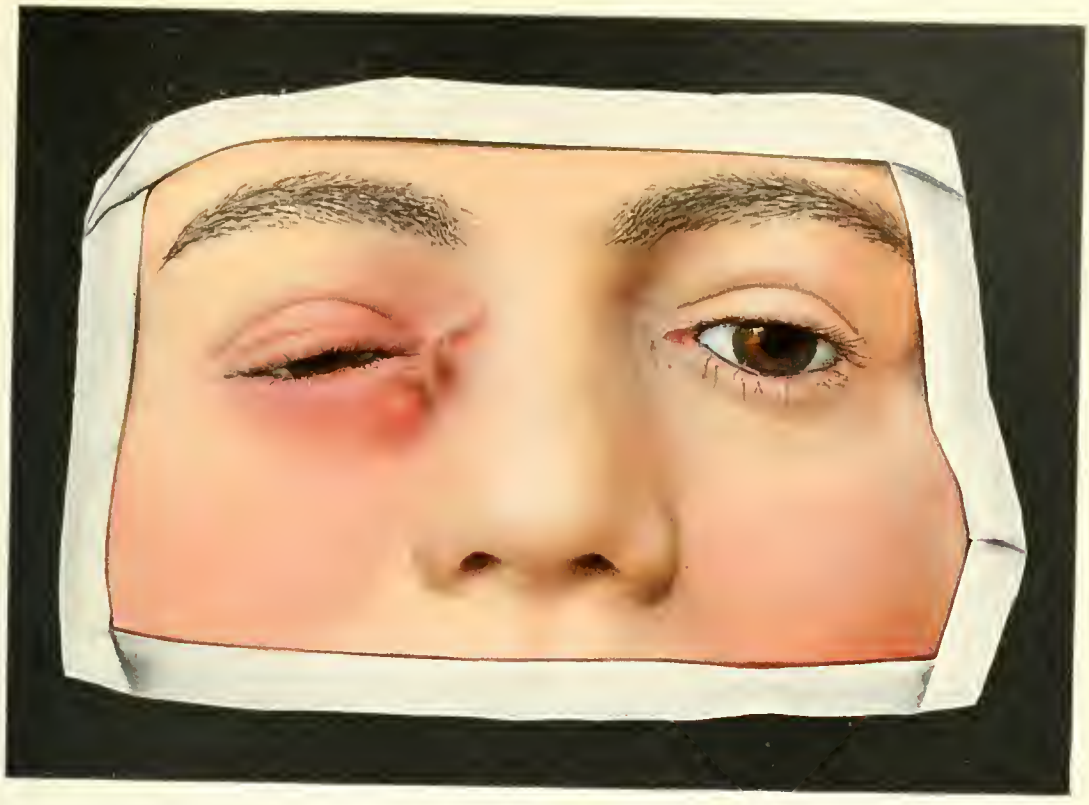

Fig. 22. Dacryo - Cystitis acuta

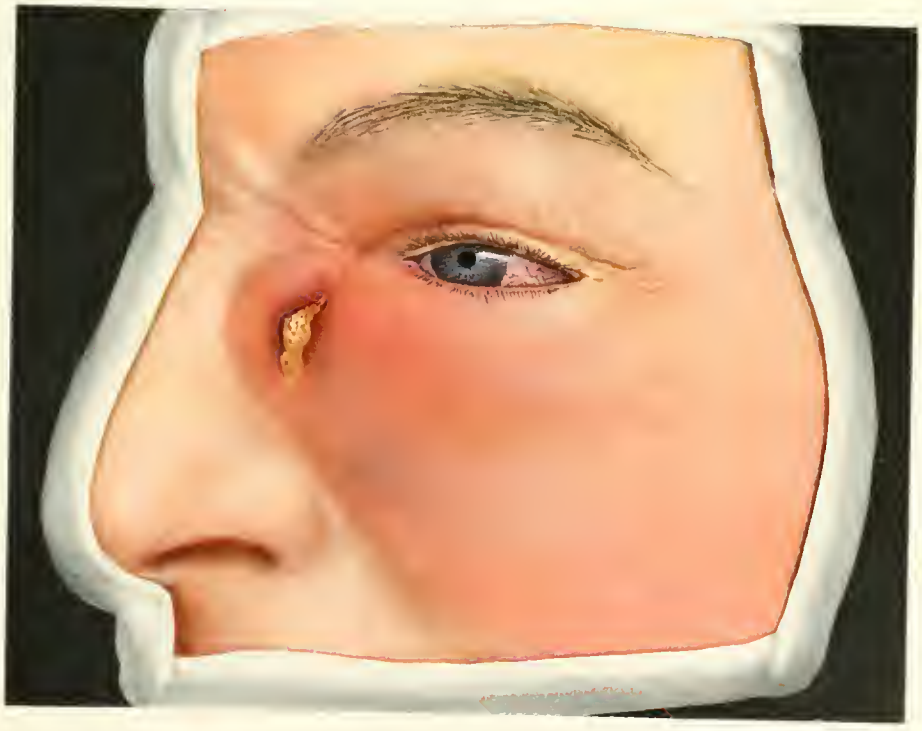

Fig. 23.
Dacryo-Cystitis with Fistula.

The extreme virulence of this pus from the lacrimal sac was known long before bacteriologic investigations were invented. It was known also that the intact eyeball might be long exposed to the regurgitant pus from the lacrimal sac without suffering noticeable injury, until some minute lesion of the cornea permitted its entrance. Then a white speck developed upon the injured superficies of the cornea, a bacterial colonization, from which an ulcus serpens had origin.

In pus from the lacrimal sac, extremely virulent pneumococci are usually found, sometimes in almost pure culture.

Such chronic lesion of the sac may also be tuberculous, tubercles or tuberculous ulcers developing in the walls of the cavity.

It is likewise frequent in chronic trachoma, trachoma follicles being found in the sac walls.

**Therapy.** This should, above all else, be directed against the often etiologic nasal lesion. To cure affections of the lacrimal sac and duct, the pus in the sac must be frequently evacuated by pressure with the finger. Then the superior or inferior punctum lacrimale must be slit or enlarged (with a Weber knife) and, after the introduction of an Anel syringe, irrigation, first with disinfectant and then with astringent solutions, should be practiced, the fluid running through the nose and into the mouth. If these injections show that the ductus naso-lacrimalis is permeable, but constricted in places, Bowman's sounds may be used in gradual dilatation.

If the duct be completely obliterated or if the use of the sounds be ineffectual, the radical operation or removal of the sac is indicated.

### Congenital Lesions of the Lacrimal Sac.

Not rarely, pus flows from the lacrimal sac a few

days after birth. The source of the purulency is easily overlooked, and the diagnosis made of blennorrhea neonatorum, gonococcal in etiology. This condition is often seen in children with congenital lues, and the suppuration may be caused by a neighboring osseous lesion. An antisyphilitic medication often suffices for the cure of the trouble. But there may be merely a simple retention in the lacrimal canal, an occlusion in the direction of the nose, and no real blenorrhea, in which case a single forcible pressure upon the dilated sac may open the channel and cause the recurrent pus to disappear.

### Acute Dacryocystitis.

The syndrome is quite different when, from any cause, the pus breaks through the wall of the sac, for then it quickly spreads through the loose adjacent tissues, the lids swell edematously and soon we have a simple, subcutaneous furuncle in the center of which lies the dilated lacrimal sac.

A case of this sort is seen in Plate XVIII., Fig. 22, to which the name, acute dacryocystitis, is given (anatomically, it would be called a pericystitis or cystitis with rupture).

**Diagnosis** must establish where, in the often marked and extensive tumefaction which may attack both eyelid and cheek, the induration is located, which, if pressed upon, causes pain. It is noticeable that in the region of greatest swelling there is frequently a deep horizontal furrow above and below which are two turgid red elevations (*vide* Fig. 22). The furrow is caused by the ligamentum canthi internum originating in the inner canthus, passing over the lacrimal sac and inserted into the lacrimal bone.

**Therapy.** Warning is given against the use of injections or of sounds during the stadium of inflam-

mation, the result of which would be extension of the infection elsewhwere. The lesion should be treated as a furuncle, using warm compresses and poultices and finally making a wide incision.

If not dispersed, the pus at last breaks through the external skin and flows out (*vide* Plate XVIII., Fig. 23), after which the lesion usually heals. If the suppuration still continue, it is commonly due to caries of the lacrimal bone. The wound must then be widely opened, curetted, and packed with iodoform gauze.

After the healing up of all these processes, there may still remain fistulæ of the lacrimal sac.

# Normal Conjunctiva Palpebrarum.

Plate XIX., Fig. 24.

The conjunctiva forms a sac or bag slit open along the line of the palpebral fissure, at the margin of which it is transmuted, without demarcation, into ordinary skin. In the conjunctival sac three areas are distinguished, *viz.*: 1, the C. palpebrarum; 2, the duplication or fold, C. fornicis; 3, the C. bulbi or scleræ.

The C. palpebrarum alone has the properties of true mucosal tissue, and hence we find that mucosal lesions, *e. g.*, infections such as gonorrhea, diphtheria, etc. as well as follicular affections, develop only in the conjunctiva palpebrarum. The C. bulbi is epidermal in type, and, therefore, dermal lesions, *e. g.* eczematous, are directly bulbar in location.

In Plate XIX., Fig. 24, is seen the conjunctiva tarsi of the upper lid after eversion. The mucosa is pale and smooth and beneath it, the glistening yellowish Meibomian glands are distinctly visible.

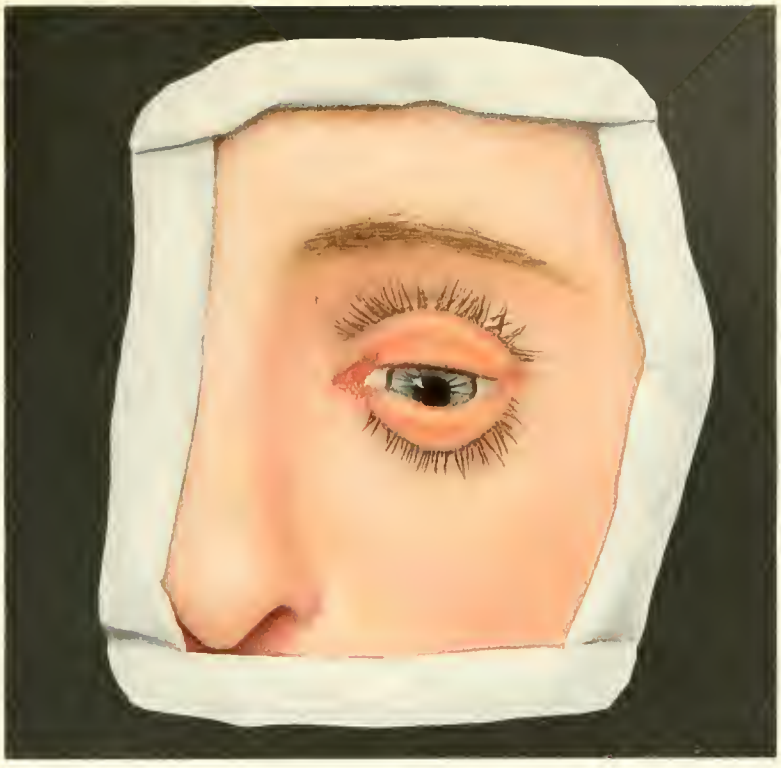

Fig. 24.
Conjunctiva. Normal Condition.

Fig. 25. Conjunctivitis catarrhalis simplex.

Rebman Company, New York.

# Conjunctivitis Catarrhalis.

PLATE XIX., FIG. 25.

The conjunctiva is a delicate, sensitive tissue, becoming hyperemic and irritated from the least stimulus, *e. g.* exposure to air, inundation with water or the salty lacrimal secretion, entropium, etc. If the irritation continue for some time or augment, there develops an inflammation of the membrane, which may exhibit the form of an acute or chronic catarrh. The surface of the swollen and inflamed mucosa does not long remain smooth; elevations and wrinklings are soon in evidence and the conjunctiva presents the appearance of a piece of finely granulated leather or clipped velvet (a papillated appearance, *vide* Plate XIX., Fig. 25), actually due to wrinkles and furrows in the tumefied mucous membrane.

If these irregularities increase in volume, become cockscomb-like, they are called papillary elevations, and are to be carefully differentiated from follicles (*vide* C. follicularis, *infra*).

Acute conjunctivitis is mostly due to an infection, whence it may properly be termed, conjunctivitis infectiosa. It is capable of transmission and sometimes excites widespread acute epidemics (*cf.* in contrast, the behavior of trachoma, often confused with this affection). Recent bacteriologic investigation has taught us that there are several species of acute, infectious conjunctivitis, also differentiable clinically; *e. g.* conjunctivitis caused by the pneumococcus, the Morax-Axenfeld diplobacillus, the Koch-Weeks bacillus, streptococci, etc.

Chronic conjunctivitis is due either to external noxæ (dust, smoke, wind, cold) acting upon hypersensitive membranes or is a sequela of acute infections.

**Therapy.** Our treatment is directed towards disinfection of the infected mucosa, and the lessening of secretion and the enormous dilatation of the blood-vessels by means of astringents.

Where secretions are dammed up, micro-organisms multiply rapidly, and nothing is more senseless than prolonged bandaging of an eye endeavoring to rid itself of such products. On the contrary, we should seek by frequent changing of compresses and by irrigation, to provide for unhindered secretion and its removal. Since the conjunctiva bears well both the action of cold and cauterizing agents, we choose cold solutions (water, lead-water, boric acid, sublimate 1:5000, etc.) in order to profit by the synchronous astringent effect of the low temperature.

Medicaments may be instilled *guttatim* into the conjunctival sac, or, after eversion of the lid a thick brush dipped into the solution may be passed over the diseased area. The following drugs are commended: zinc sulfur, ¼ to 1 or 2%; zinc sozoiodol, ½ to 1%; solution of alum 1%; sod. sozoiodol, 3 to 6%; resorcin, 1 to 2%; acid. tannic, 1 to 2%. In purulent secretions, the sovereign remedy is silver nitrate, 1/5 to 1% or its substitutes (protargol, argentamin, albargin, etc.).

Fig. 26.
Conjunctivitis follicularis.

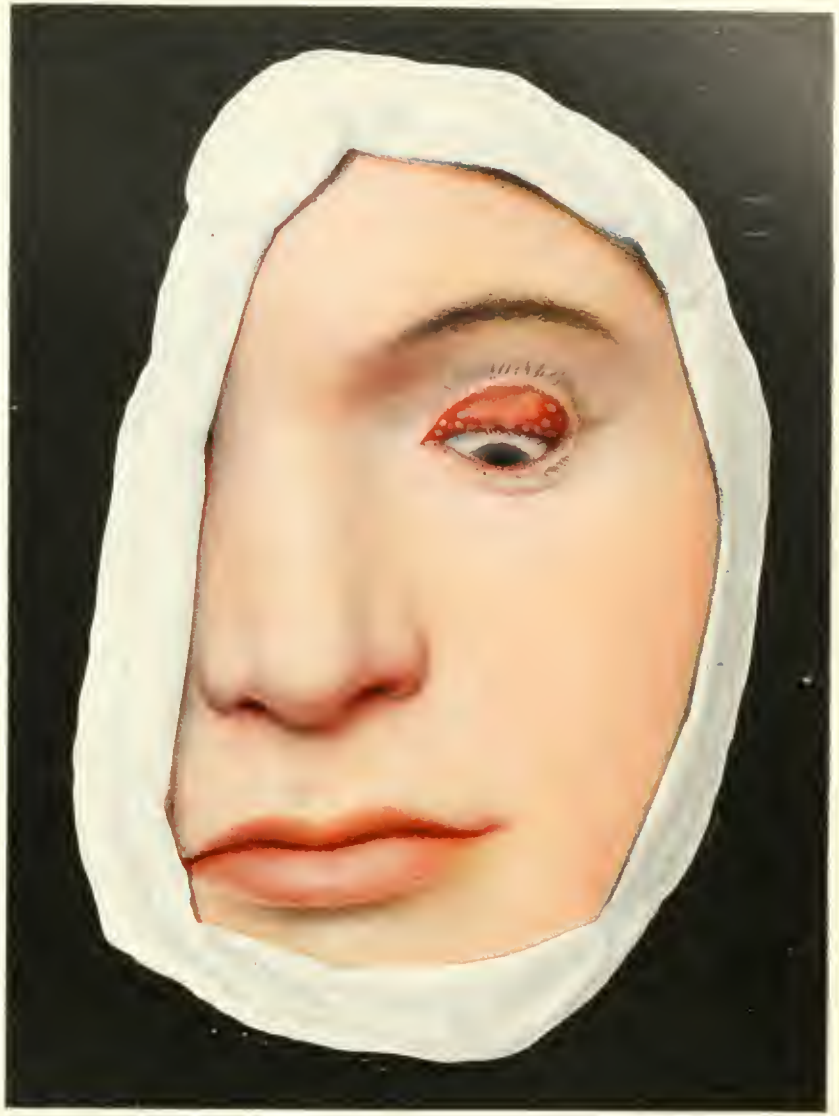

Fig. 27.
Conjunctivitis trachomatosa I.

# Conjunctivitis Follicularis.

Plate XX., Fig. 26.

Another species of elevations on the conjunctiva palpebrarum is the follicular. The follicles at first resemble transparent vesicles; later, and in malignant forms, they look like frog-spawn or cooked sago grains, and are composed of circumscribed aggregations of round cells underneath the epithelium. They are, therefore, neoplasmic, comparable to lymph follicles or lymphomata, and develop in the conjunctiva from the most varied stimuli (chemical, thermic, bacteriologic). On the other hand, it must be emphasized that not every stimulus or irritation develops these follicles, nor are they due to the intensity or prolonged action of the irritation but to its specificity. Simple chronic catarrh, however long its duration, never develops follicles; they are also absent in the most violent conjunctival inflammations we know, *viz.* gonococcal or diphtheritic infections. Contrarily, the formation of follicles is noted in a large number of conjunctival affections, plainly due not to infection alone, but also to other irritations. The various species of conjunctivitis may, therefore, be divided into those beginning with the development of follicles, and those where such formation is absent.

A certain percentage of children with conjunctival follicles are found in all schools, but the conjunctiva is pale in color and normal and the condition causes no trouble. In such cases, diagnosis of conjunctivitis follicularis is incorrect, for there is no conjunctivitis.

The chlorotic and anemic children have merely dilated lymph vessels in the conjunctival superficies,—lymphectasia. Follicles develop whenever numbers of individuals are crowded together in a heavy, impure air, as in overfilled public schools, in schoolrooms especially, in orphan asylums, etc. All inmates of penitentiaries and prisons show, post mortem, microscopic follicles in the conjunctiva. That in such cases symptoms of conjunctival inflammation are usually absent, is due, in my opinion, to the fact that, as a rule, it is not a question here of infection, the phenomenon being due to the irritant atmosphere, some authorities attributing it to the ammoniacal vapors, others to the anthropotoxin present, etc.

There are, however, plainly infectious but mild conjunctivites in which follicles appear, but, generally, not numerous, superficially located and chiefly in the lower lid. These follicles disappear without leaving scars.

**Therapy.** Sojourn in a pure atmosphere and exercise in the open air often suffice. The follicles so often found in school children frequently disappear spontaneously during vacation. In addition, the eyes should be repeatedly washed, and compresses of 2-4% boracic acid solution or leadwater applied. For instillation, Förster's 2% solution of sod. biboracicum is best.

Fig. 28. Conjunctivitis trachomatosa

Fig. 29. Sklerosing Trachoma.

# Conjunctivitis Trachomatosa.

Plate XX., Fig. 27; Plate XXI., Figs. 28-29.

Trachoma is an infection locating in the mucosa of the lid, and is indubitably a specific morbid entity, whose cause we do not know with certainty. It is not communicable to animals, though possibly transmissible in mild form to the anthropomorphous apes.

Trachoma is distinguished by the formation of large follicles which, accompanied by inflammation and marked papillary tumefaction, constantly increase in size and number and finally lead to necrosis and subepithelial shriveling.

It is a world-wide disease, but its occurrence is not symmetric, *i. e.* in regions free of trachoma are found here and there larger or smaller areas in which cases of trachoma are always numerous, so-called "trachoma islets."

Is it possible to differentiate with certainty trachoma and follicular catarrh? In most cases, yes, though at first not always without error, though diagnosis may be invariably established by more extended observation. In follicular catarrh, we have absolutely benign new-formations, usually with no adjacent inflammation, no reaction, no tumefaction, and which do not lead to any notable swelling of fornices and papillæ, and which, in contrast to trachoma, preferably develop in a pale, anemic mucosa. Their lack of malignancy is shown by the fact that they never lead to destruction of mucosal tissue, but sooner or later disappear spontaneously, leaving no trace of their existence.

It is very different with the trachoma granule. It generally presents an entirely different appearance, for, whilst the follicular granule is clear and pale, more like a vesicle, the deeper-seated granule of trachoma soon becomes gray and discolored. The circumjacent mucosa is deeply reddened, tumefied (so that the trachoma granule seems to lie deeper below the surface) and the swollen fornices, when the lid is everted, roll out as if enlarged and thickened. Very soon there develop notable papillary proliferations, indicative of the violent reaction excited by the powerful virus—phenomena always absent in follicular catarrh. In its course, the malignant character of trachoma is made perfectly evident. The trachoma granule does not, after existing for some time, return to a norm, but is distinguished by its destructive action upon the mucous membrane. It tends notably toward a metamorphosis of the mucosa into a tough, cicatricial tissue and develops a sort of cirrhosis, similar to that caused in lung, liver, kidney by certain inflammatory processes, and because of this we get the common sequelæ of trachoma. The cornea is often implicated and pannus develops.

Trachoma is at the present time a pre-eminently chronic disease, endemic in many regions, absent in others. When endemic, it pursues its course, and, probably has for centuries; one patient after another is slowly infected, and, of a family of six, possibly three are trachomatous, and, gradually, after months or years, the others will be. The transmission is not easily accomplished, and continual, prolonged association with those infected, a common use of sleeping places, towels, etc., are requisite. If an individual is infected, it is often a long time before he notices the slightest sign of inflammation, and it may be months or years ere the process becomes malignant. Some

times, however, the disease, in rare and isolated cases, runs a violent course. If a patient with bad trachoma enter some institution, he usually infects, after some months, a greater or less number of his comrades, and, in this manner there gradually develops a small endemic. But, I have never found all the inmates of such an institution affected, which is proof that even from long and daily association infection need not follow. Trachoma, therefore, is always present in regions where it is indigenous; one individual after another is slowly infected, whilst other cases recover, so that the number of those diseased varies within certain limits; some years perhaps less, other years, greater. But, the report that a sudden outbreak of trachoma has occurred, has invariably been proven false. Either it was not trachoma, but rapidly spreading innocuous catarrh accompanied by tumefaction, or some other disease, or else in the regions where trachoma had existed for years, attention has been suddenly fixed again upon the disease.

As insidious and chronic as is the progress of trachoma from patient to patient, so is its course, which if not interfered with, may extend over years or even a lifetime. Quite a number of cases finally recover. But even the best treatment must cover months, and be extremely energetic, if a sure and permanent cure is to be attained. Relapses are very frequent. The disease is one of the most malignant ophthalmic diseases known, and, in common with blenorrhea neonatorum influences largely the number of the blind. Still greater is the number of patients who, while not entirely blind, suffer greatly in later life from its sequelæ.

What happens, finally, to the trachoma granule?

After long duration, there develops always in its interior a sort of softening, and we find the large cells

in the stadium of necrosis, the nuclei no longer staining, until at last there is left only a granular, crumbly mass in which the original elements have vanished.

In many cases, the softening does not develop equally throughout the contents of the follicle but begins in foci, but commonly the central portion is most affected.

The most typical illustration of the softened follicle is the clinical picture of the so-called gelatinous trachoma (Stellwag). Here the follicular formations stand so closely together that the single, softened follicles merge into one jelly-like mass (Fig. 29).

According to Rählmann, Addario and others, the rupture of the follicle and exudation of its contents is the natural termination.

In my opinion, the gradual resorption of the follicular contents is by far the more frequent and natural process. This resorption is found, apparently, not only when the contents are softened, but according to clinical experience, in any stadium. Thus, we have seen minute follicles, where there can be no possible softening, gradually disappear in numerous instances when treated medicamentally, in which cases there was certainly no rupture and exudation of the contents.

For the **therapy** of trachoma, consult pp. 69-70.

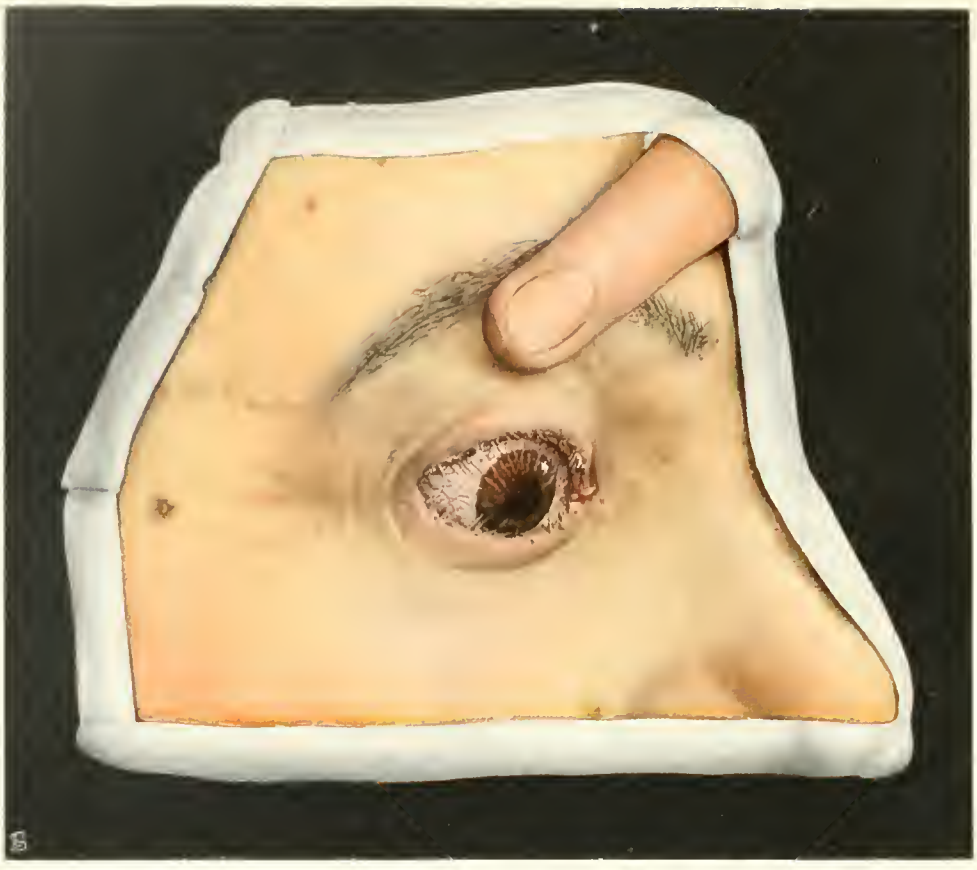

Fig. 30. Pannus trachomatosus.

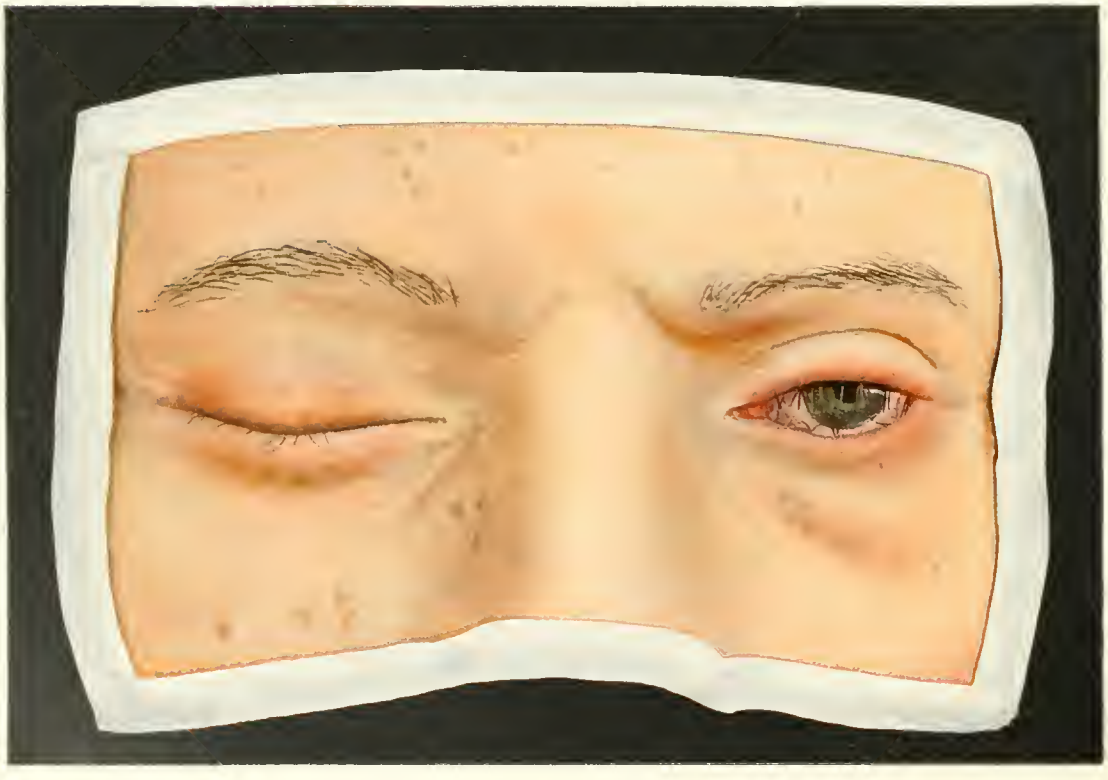

Fig. 31. Entropium and Trichiasis through scar-tissue.

# Pannus Trachomatosus.

Plate XXII., Figs. 30, 31; Plate XXIII., Figs. 32, 33.

The trachomatous process may pass from the conjunctiva palpebrarum et fornicis to the cornea without attacking the conjunctiva bulbi, and upon the cornea develop the pannus trachomatosus, starting almost invariably at the corneal margin and usually from the upper portion. Here are seen at first, small, circumscribed elevations demonstrable only with a magnifying glass,—minute but distinct points rising above the corneal surface, sometimes attaining the size of a poppy-seed. These solid nodules, gray-white in color are follicles, circumscribed, subepithelial infiltrations of clearly defined masses of lymphoid cells. If the nodules already lie in the transparent corneal tissue, many of them may be seen surrounded by slightly cloudy areolæ. Later, the nodules become confluent, forming a soft, diffuse mass, rich in cells, which, subepithelially, push forward from the superior margin of the cornea across its transparent tissues. Since this neoplasmic layer is not everywhere of equal thickness, the overlying epithelium is humped up here and there. As soon, however, as the layer has progressed a millimeter or more across the transparent cornea, there begins, at its superior edge, a vascular proliferation extending with it across the cornea but always somewhat posterior to the zone of infiltration. These blood vessels do not all extend meridianly toward the center of the cornea, but are inclined rather to run in parallel from above downward.

According to the vascular development, the pannus varies in appearance. A fresh pannus, with few of these blood vessels extending downwards, is called *pannus tenuis;* if they are numerous, *pannus vasculosus.* Sometimes, the new tissue becomes so thick and vascular that it appears like granulation-tissue or raw flesh lying upon the cornea, and is then termed *pannus crassus* or *carnosus;* less suitably, *pannus sarcomatosus.*

With retrogression of the growth, the zone of infection first recedes and the blood vessels follow the vascularity, thus always remaining longer in evidence (*vide* Fig. 45). Because of this, the progressing and retrogressing panni are always easily differentiated. As soon as the pannus and the blood vessels have somewhat passed the central point of the cornea, the picture changes. The blood vessels no longer run parallel from above but extend in all directions on the corneal surface, in whose central portion they frequently anastomose and form varicose swellings (*vide* Fig. 46), and it is seen from their more indistinct, bluish hue, that they now lie, in many areas, deeper in the tissues. An old pannus, with connective tissue metamorphosis, has usually but few blood-vessels and these of diminished caliber: *pannus siccus.*

### Cicatricial Trachoma.

Finally, and commonly after persisting for years, the trachoma leads to a more or less extensive cicatricial contraction of the affected mucosa. The tarsus is, as a rule, implicated later. Its tissues, at first densely infiltrated, exhibit marked tumefaction, but, in time contract, and we have a rigid, sclerotic involution (Plate XXIII., Fig. 32).

The tarsal distortion is typical in all cases, *i. e.* there is not equal curvature of the cartilage but a percep-

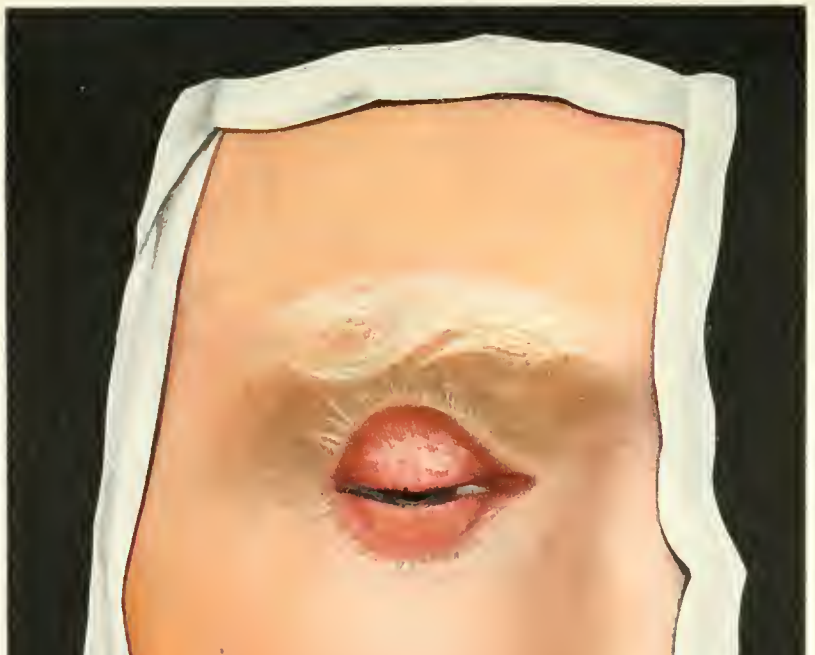

Fig. 32.
Trachoma cicatriceum.

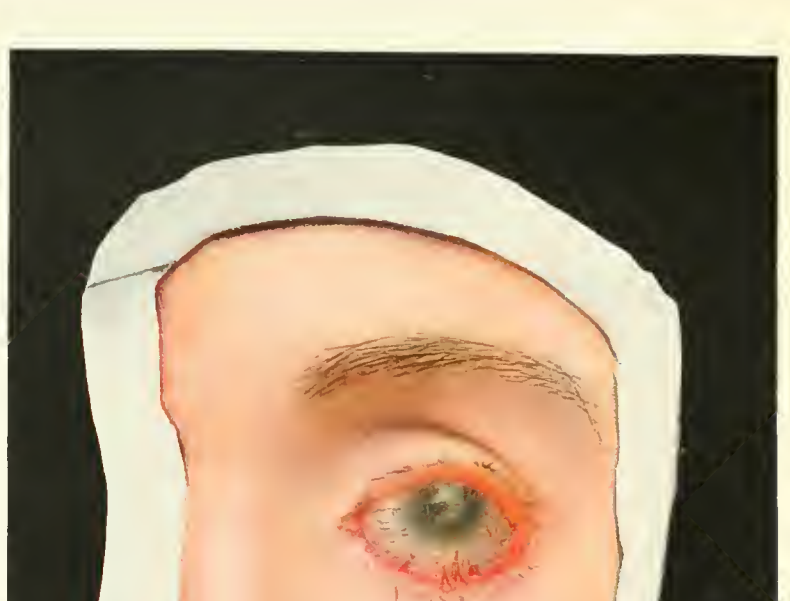

Fig. 33.
Xerophthalmus trachomatosus

tible notch in its middle portion. It is easily cognized that the callous thickening of the conjunctiva is greatest at a point corresponding to this notch, so that here, in a fashion, the punctum fixum of the cicatricial retraction is located, because of which the two cartilage margins are displaced by the retractive action of the scar. Hence, the distorted, trough-like cartilage is, because correspondent to the thickest portion of the callous conjunctival cicatrix, more or less indented.

With the inward bending of the tarsus, the free margin of the lid is also bent inwards, and there develops entropium with its well known grave sequelæ (Fig. 31).

If, finally, the conjunctiva becomes so cicatrized, that the cornea is no longer sufficiently moistened, the entire surface of the eyeball becomes rough, dry, epidermoid, a condition called xerophthalmus (Plate XXIII., Fig. 33).

The **therapy** of trachoma. We have medical, mechanical and surgical methods of treatment. Of medicaments, two are pre-eminently useful: silver nitrate in 2% solution in new cases, particularly where the secretions are copious, applied with a brush or swab; and copper sulfate where the resorption of follicles is desired. The latter is best used in the crystal form, the so-called blue-stone or pencil, with which the entire conjunctival surface is gently and equably stroked once daily. Cold compresses are then applied, the use of the stone being discontinued, if the ophthalmic condition exhibits progressive irritation. If the patient is not seen daily by the physician, instillation of a ¼% solution of copper sulfate twice daily is prescribed, or it may be used in the form of a 1% unguent. Many physicians prefer Arlt's use of copper citrate, usually as unguent, and now obtainable, under the name of

cuprocitrol, in tubes of 1-5 grams, from the Schürer v. Waldheimschen Apotheke in Vienna. With a glass rod some of this is rubbed once daily over the conjunctival surface. It is less irritant than other applications, and, therefore, safer for the patient's own use.

Iodine preparations are also frequently employed: Tinct. iodi, 1.0, glycerine, 15.0 as collyrium; iodoform as dusting-powder or in unguent; pure iodine (½-1%) dissolved in glycerine and daubed with a cotton application over the everted lids, or, a caustic pencil may be made of pure iodic acid moistened with a very little water until plastic, when it may be rolled on a glass plate into pencil form, and with this, cauterization may be performed about every third day. The pain is intense, but soon passes off.

Of the mechanical procedures, that of Keinig is worthy of mention. A cotton applicator is dipped into a 1:3000-5000 sublimate solution, and the cotton rubbed vigorously over the diseased mucosa, repeating this every second day until the granules have disappeared.

Operative methods have the advantage of greatly shortening the duration of the disease, a desideratum in epidemics. But, not all of them are radical, and it is always advisable to follow with medicamental treatment. With Knapp's roller-forceps, built on the plan of a clothes-wringer, the new-formed trachoma granules may be expressed. In older, deeper infiltration, excision of the fornices or of the tarsus is recommended.

Trachoma is infectious, and preventive measures are, therefore, to be prescribed. The most dangerous carriers of the contagium are dirty wash-water and towels.

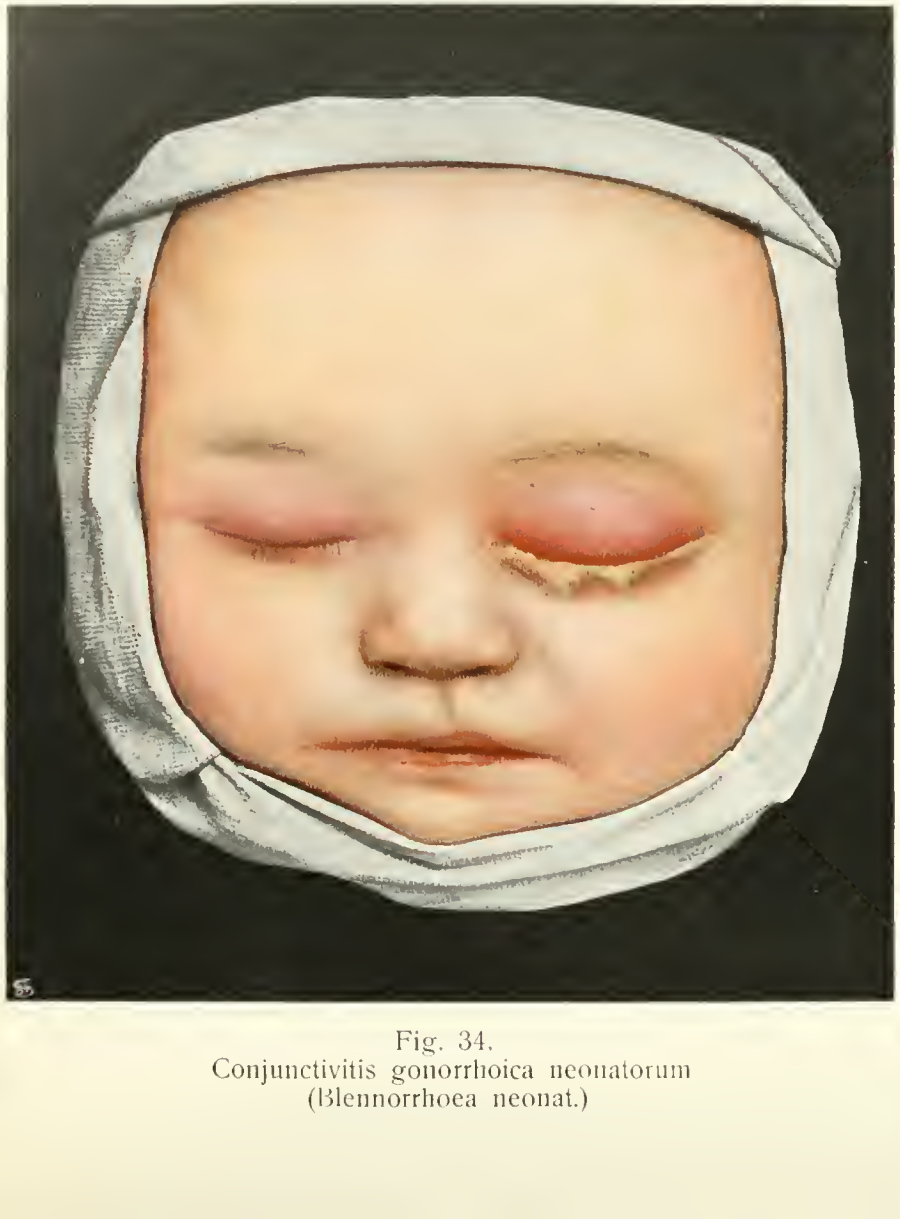

Fig. 34.
Conjunctivitis gonorrhoica neonatorum
(Blennorrhoea neonat.)

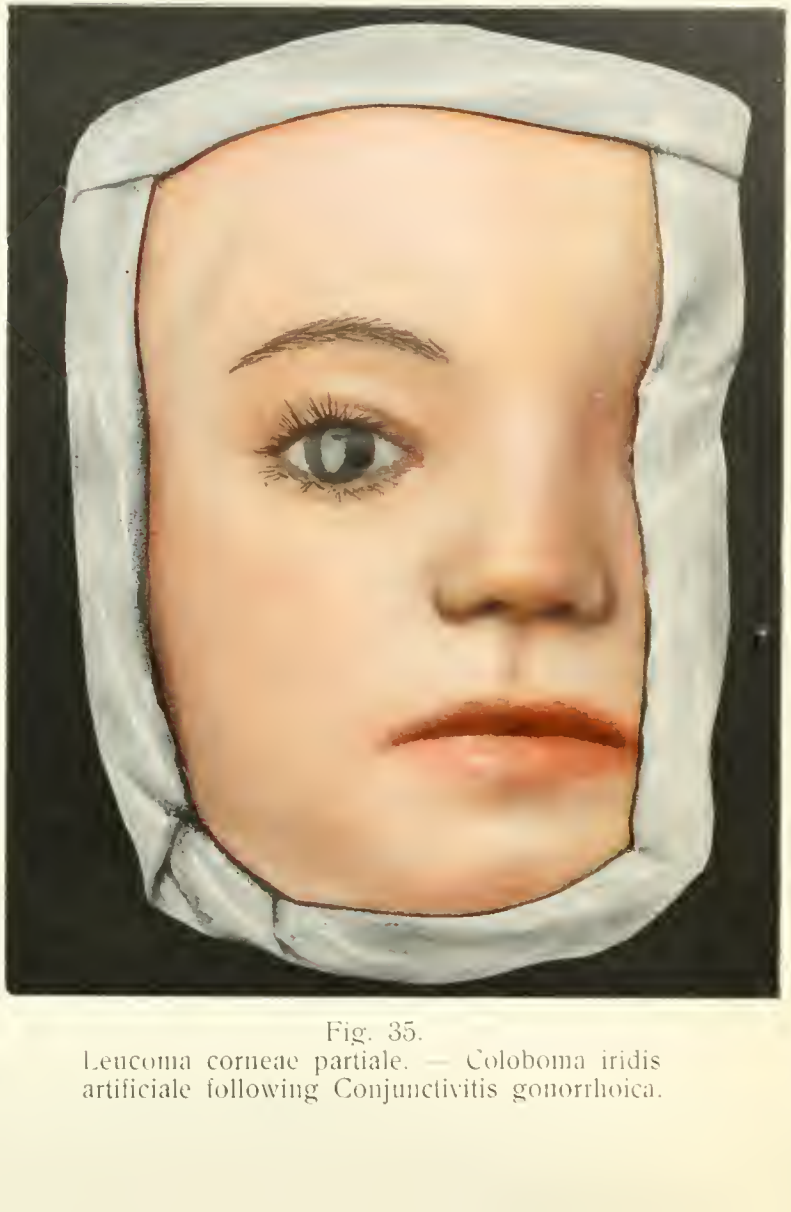

Fig. 35.
Leucoma corneae partiale. — Coloboma iridis
artificiale following Conjunctivitis gonorrhoica.

# Conjunctivitis Gonorrhoica.

Plate XXIV., Figs. 34-35; Plate XXV., Figs. 36-37; Plate XXVI., Fig. 38.

Gonorrheal disease of the eye is chiefly caused by infection from the exterior, a so-called gonorrhea by contact, but, we have other types, and these ophthalmias may be classified as:

1. Gonorrhea by contact:
2. Gonorrhea by metastasis from other foci in the body.
3. Gonorrhea where metastasis occurs to other parts of the body, especially the articulations, nowadays so much observed and investigated by the surgical staff.

Furthermore, ophthalmic affections due to a contact gonorrhea are often divided into conjunctivitis gonorrhoica neonati and hominis adulti, remembering, however, that there is no qualitative difference betwixt them. Differentiation is, perhaps, advisable, first, because of origin, secondly, because of prognosis.

### Conjunctivitis Gonorrhoica Neonatorum.

Ophthalmic gonorrhea following birth is due to infection from outside: a so-called contact-gonorrhea.

Neisser's gonococci enter the conjunctival sac during birth and often, because of lack of cleanliness, after birth. After brief incubation, varying, with the severity of infection, from a few hours to days (commonly after 2-3, more rarely, after 4-5 days), the disease breaks loose. The lids become very red and swollen, but chemosis is rare. Out of the palpebral fis-

sure flows a secretion at first turbid, then wholly purulent, in which, lying within the leucocytes or desquamated epithelia, are found, usually in pairs and very numerous, the gonococci, not staining by Gram's method. After some weeks the thick, creamy pus becomes thinner, and at last ceases. There is no chronic conjunctivitis gonorrhoica. When there is stasis of the pus it generally corrodes the corneal epithelium, which then, in greater or less measure, disintegrates by suppuration. After cession of the inflammatory phenomena, the defect is hidden beneath an opaque, whitish cicatricial tissue (Plate XXIV., Fig. 35). In Fig. 35 in the upper part of the leucoma is a small black line, an anterior synechia, *i. e.* after slight perforation of the cornea, a portion of the iris has fallen forward and through and healed fast (*vide* also Fig. 55 on Plate XXXVIII., leucoma corneæ totale, where the destruction of the cornea was due to variola, exactly similar in appearance, however, to that caused by conjunctivitis gonorrhoica.) If the cornea be totally destroyed by suppuration, a protective partition is formed by the contraction of the freely exposed iris across the pupillary area (*vide* Plate XXVI., Fig. 38), but later, intraocular pressure drives this curtain forward and we have a total staphyloma corneæ (*vide* Plate XLI., Fig. 59).

If pus find its way into the interior of the eye there develops, after long and violent inflammation, a gradual wasting of the eyeball: phthisis bulbi (*vide* Plate XXV., Figs. 36-37).

The conjunctiva is markedly red and swollen, the papillæ spring up in ridges or cockscomblike, but there is never formation of follicles (as in conjunctivitis follicularis or in trachoma), nor, after the disease has run its course, do we find cicatrices in the conjunctival tissues.

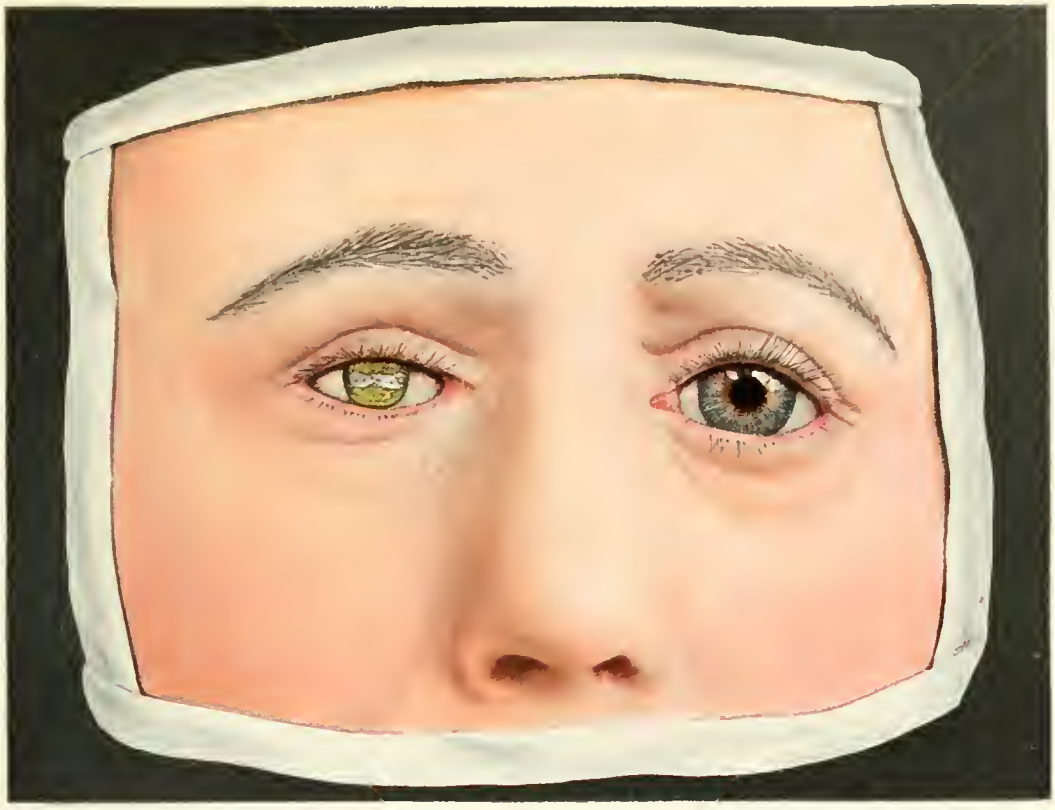

Fig. 36. Phthisis bulbi incipiens.
Adhesion form of Keratitis.

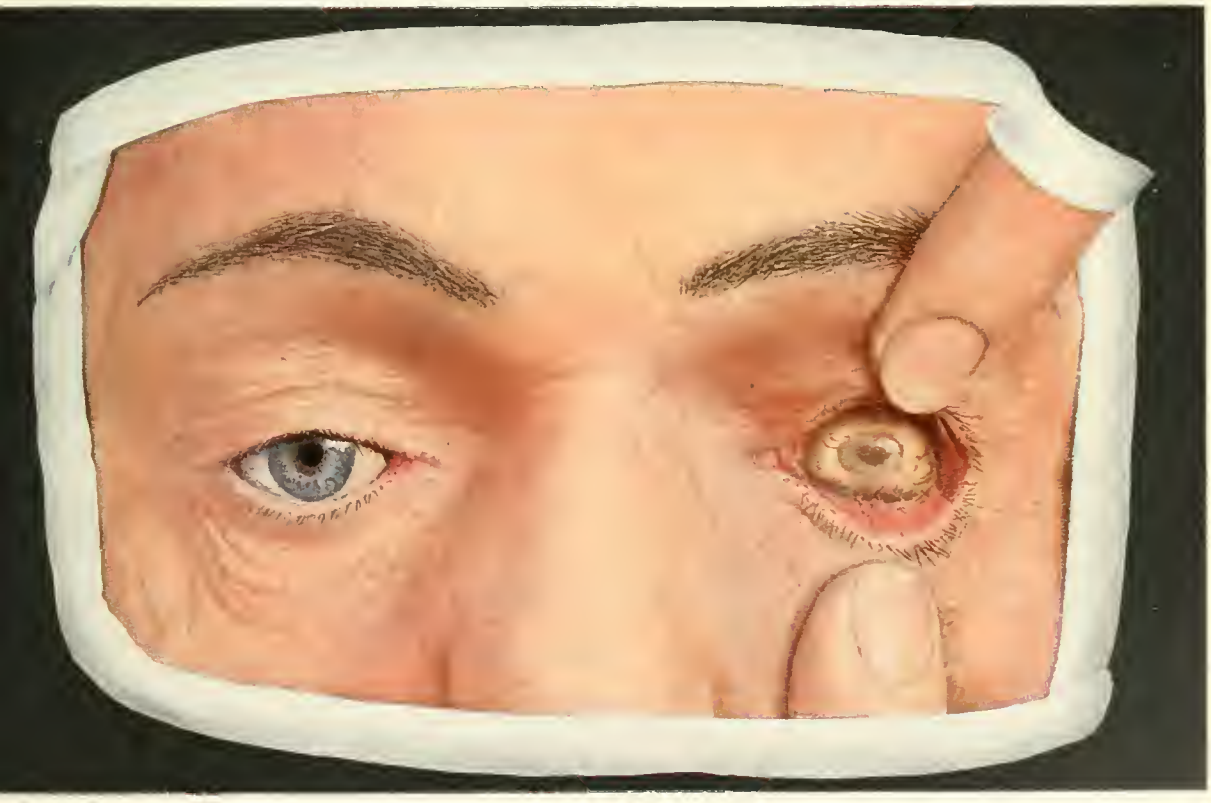

Fig. 37.
Phthisis bulbi quadrata.

One might suppose that the eye of the adult was but slightly sensitive to the gonococcal virus, and, apparently, the supposition is supported by statistics. In the large cities gonococcal urethritis is extraordinarily common, but the percentage of ophthalmic infection is extremely small, perhaps not one in a thousand individuals with urethritis gonorrhoica are ocularly affected, so that it would seem true that the eye has very slight receptivity. And yet the cases where the eye has actually become infected disprove this. This is due to the protection afforded the eyeball, making infection difficult, and to the fact that the gonococcus is a very delicate and easily destroyed microorganism.

The fresh pus must be brought directly into the eye, and even rubbing with the infected hand does not always infect the eye, for we are not in the habit of touching conjunctival tissues, and on the derm, on the skin of the lid, the gonococcus finds no point of attack. At any rate, cases where the virus has actually got into the eye, show its extreme sensitivity to the gonococcus.

Conjunctivitis gonorrhoica in adults begins violently, much more so than in the newborn, and it is important to know this. The numerous patients with a urethritis gonorrhoica, and who know that the disease is transmissible to the eye, are in constant anxiety. If it happen that a few reddened blood-vessels show in the eye, such a patient, naturally, is greatly disturbed, for he suspects infection. Such individuals are very numerous in the consultation room. And, when we see a conjunctivitis with marked redness, we can usually tell from its period of duration whether it be gonorrheic or not. Gonorrheic phenomena do not begin with redness and inflammation of the con-

junctiva, but with extremely violent lacrimation, with hourly aggravation of the symptoms, so that if in the morning the tears were clear, at midday they are turbid, and by night the gonorrheal picture, a wave of pus rolling from the eye, is in evidence. When we learn, therefore, that the symptoms were present yesterday or the day before, and still the characteristic syndrome of a gonorrheic ophthalmia is not before us, we may usually exclude an infection and pacify the patient. When such syndrome develops, we have the marked swelling and edema of the lids, the conjunctival chemosis, and the specific trickling of pus from the conjunctival sac.

In adults, the cornea is much more apt to become affected than in the newborn, so that even the most careful treatment is not always able to hinder an unfortunate termination.

### Conjunctivitis Gonorrhoica Metastatica.

Besides the above-described conjunctivitis from contact or direct infection, we have a conjunctivitis gonorrhoica metastatica, somewhat frequently observed in modern times. As we have learned to recognize affection of the joints as, not rarely, metastases of gonorrhea, so the ophthalmic metastasis has been often noted. The eye is peculiarly apt to metastases.

The picture of ophthalmia gonorrhoica metastatica is entirely different from that induced by contact or direct infection. We do not have the marked suppuration; we see merely a conjunctivitis with some circumjacent edema, and often an iritis, differing little from any other iritis. In the great majority of cases also, there are no gonococci found in the secretions. Apparently, we are dealing with a mixed infection, for we know that the plastic inflammations often present in precisely this form of iritis are not due to gonococci.

In short, we have in the eye a number of symptoms found also in gonorrheic affections of the joints.

Finally, there are cases in which metastases to various parts of the body have proceeded from gonorrheic affections of the eye.

The **prognosis** of conjunctivitis gonorrhoica neonatorum is favorable when the treatment is correct; and the morbidity is almost always removed without sequelæ. Without treatment, the stagnating pus erodes the cornea and more or less destroys it.

In adults, the result is not so certain, for even most careful treatment does not always prevent corneal lesion.

Prophylaxis plays a chief role. Adults with gonorrheic urethritis or a gleet are to be warned against touching the eyes, and the hands should be thoroughly cleansed with soap and water after each contact with the pus. With the newborn, in suspicious cases, Credé's method of instilling a drop of 2% solution of silver nitrate into each eye is to be recommended, or better, this too strong solution may be reduced to ¼ per cent. Water used in bathing must not come in contact with the eyes.

**Therapy.** Where but one eye is affected in adults, the sound eye should be protected by a watch-glass or a piece of mica held in place peripherally by adhesive plaster. In treatment, silver nitrate or one of its substitutes, protargol or argentamin, is best. The classic method of von Gräfe's school was a daily painting over with a 3% solution, but I prefer a weaker ($^1/_{10}$-$^1/^5$%) silver solution with which the eye is frequently irrigated during the day and the pus washed out. In this procedure, all friction must be avoided, for the cornea may be easily injured (particularly by unskilled hands), with serious results. A piece of

cotton wet with the solution, or a vial with slit cork should be held at a distance from the eye and the solution allowed to drip in betwixt the lids which are simultaneously opened and closed, thus washing out all pus. Where the lids are much swollen ice-compresses may be applied for one or two days only.

To avoid maceration of the cornea by the pus, many authors commend one application daily of unguent. Recently, the 5 or 10% Lenicet ointment (Dr. Reiss, Berlin) in original tubes, or Euvaseline (Reiss) in original packages has become quite famous.

Against leucoma corneæ, all therapy is ineffectual. If partial and in the center of the cornea, one may, by excision of a side of the iris (optic iredectomy) together with the cicatricial turbidity, form an artificial pupil (*vide* Plate XXIV., Fig. 35). For a better cosmetic effect, the white spot may later be tinted black with Chinese ink.

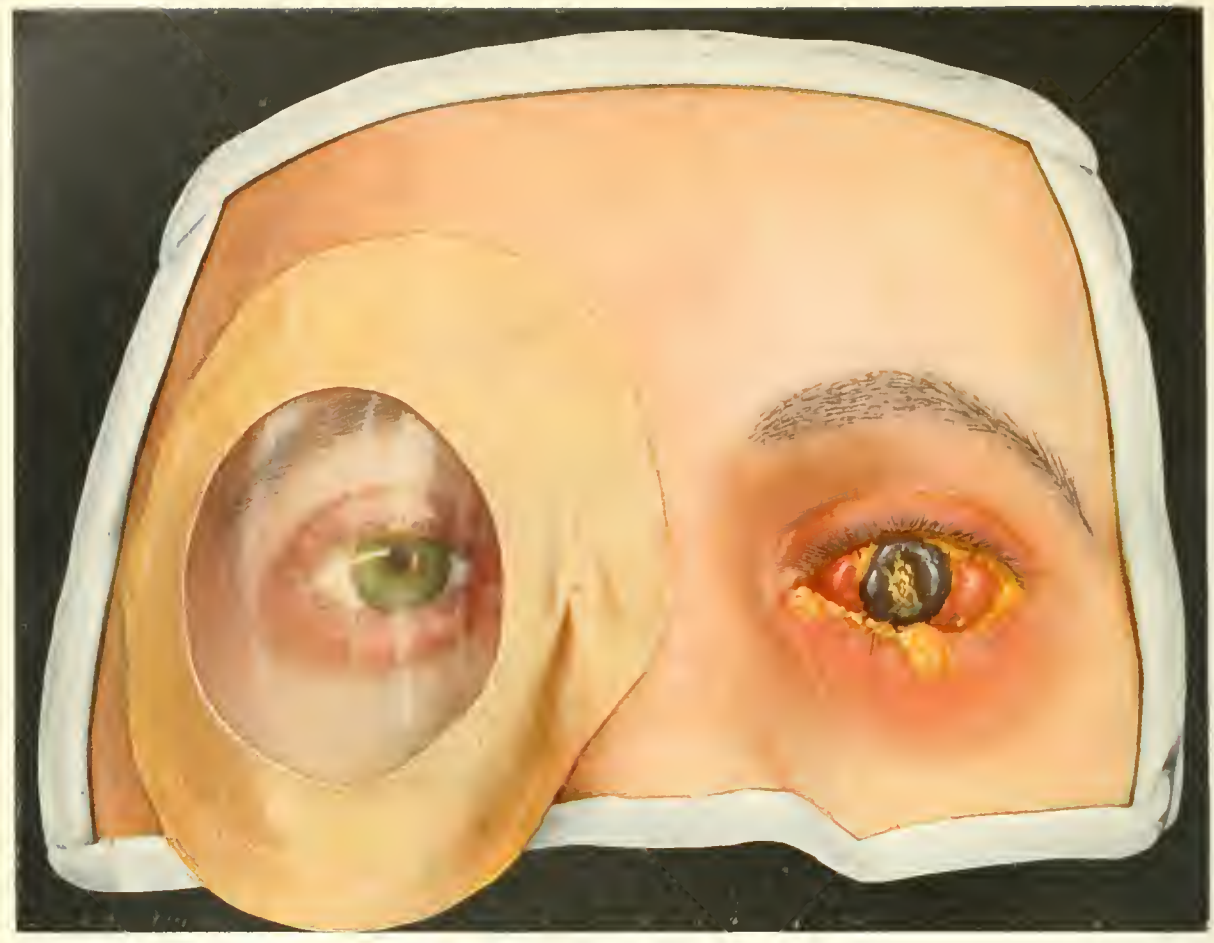

Fig. 38.
Conjunctivitis gonorrhoica adultorum.
Prolapsus iridis totalis.

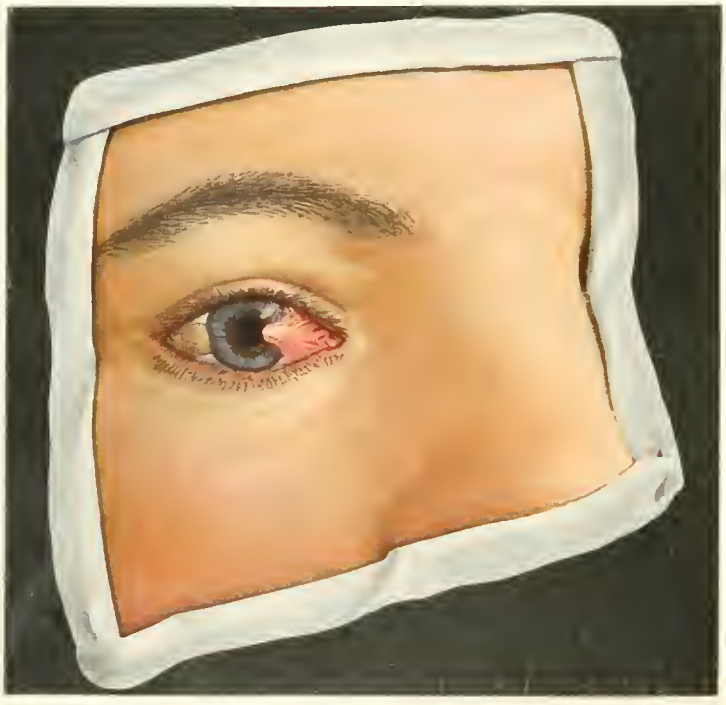

Fig. 39.
Pterygium und Pinguecula.

ebman Company, New York.

# Pinguecula, Pterygium.

Plate XXVI., Fig. 39.

Pinguecula (Lidspaltenfleck) belongs with the senile degenerations. In the region of the triangular portions of the conjunctiva bulbi lying on each side of the cornea when the eye is open, there develop in time thickenings of the conjunctival tissues which gradually become straw-colored elevations. Their evolution is favored by weather disturbances, living in an atmosphere of smoke and dust, by a hot, sunstruck climate, etc.

As the name denotes, it was formerly believed that the pinguecula was composed of fat (*pingue*, fat), but it is actually a hypertrophy of elastic tissue associated with granular pigment.

**Prognosis.** The growth has no significance, and merely disfigures. Sometimes its appearance greatly terrifies the patient or his family, and hence it is important to recognize the nature of the phenomenon. It needs no therapeutic intervention.

From pinguecula, a pterygium may develop.

Pterygium is a triangular fold of mucous membrane, growing horizontally from either side of the conjunctiva bulbi toward and over the cornea. The blunt apex of the triangle lies in the transparent cor nea to which it is firmly and immovably attached.

In a pterygium, we differentiate the head or blunt apex, then the thin neck, and, finally, the broad body lying upon the sclera.

It grows gradually and without inflammation across the cornea and is rich in blood-vessels. Usually, it is progressive and in the course of years may reach and pass beyond the center of the cornea. It is found in elderly individuals and is more common in hot climates than with us. Pterygium evolves from a pinguecula. The latter begins to grow suddenly toward the cornea, and pulls after it a flap of conjunctival tissue.

**Prognosis.** Pterygium causes visual disturbances as soon as it enters the corneal field and when it covers the pupil entirely, the eye becomes practically blind, though able to cognize light. As, commonly, it is progressive, the prognosis is unfavorable unless the growth be removed by operation.

**Therapy** comprises the ablation of the growth and suturing of the conjunctival wound. This should be done as early as possible, for the corneal area affected by the ablation will never regain its smooth surface.

Hence, after the removal of far-advanced pterygium, there always remain disturbances of vision.

Fig. 40.
Conjunctivitis diphtherica.

# Conjunctivitis Diphtheritica.

Plate XXVII., Fig. 40.

Infection of the conjunctival tissue with the Klebs-Löffler bacillus we term conjunctivitis diphtheritica.

Von Gräfe was acquainted only with the grave, deeply penetrating, necrosing form of ophthalmic diphtheria, which usually begins with corneal implication. Later, a mild, superficial form, accurately differentiated, however, from the true form, was described as croup of the conjunctiva. These nosologic viewpoints were altered only by the discovery of the active cause, for in 1886 the diphtheria bacillus was first demonstrated by Babes in conjunctival diphtheria (Prager med. Wochenschrift, 1886, Nr. 8).

Still later, the bacillus was also found in the mild, superficial form. To-day it is certain that both forms should be classified under the head of conjunctivitis diphtheritica. We must, therefore, in every conjunctivitis with superficial, pseudo-membrane and of benign course, harbor the suspicion that we are dealing with diphtheria. I say, "harbor the suspicion," for we cannot be clinically sure without a bacteriologic examination, for gonococci and, even more frequently, streptococci may form membranes. And, conversely, virulent diphtheria bacilli may develop merely a simple conjunctival catarrh (simple catarrhal form) without the formation of a membrane. We thus see the importance of bacteriologic examination.

Why the virulent bacilli develop now a benign, superficial morbidity, and again, one deeply penetrating and necrotogenic, we do not know.

In most ophthalmic diphtherias, the diphtheria bacilli are not found in pure culture, but mixed with staphylo- and streptococci, and finally, with avirulent xerosis bacilli (pseudo-diphtheria bacilli). It appears, however, that these mixed infections have no influence upon the course of the disease.

**Diagnosis** in the superficial form is not easy, for staphylo- and streptococci as well as slight cauterizations occasionally develop membranes. Demonstration of Löffler's bacillus renders the diagnosis certain.

**Prognosis** is always dubious. Many cases recover unharmed, but even the mildest type may suddenly become malignant, destroying the cornea, developing gangrene of the lids, or, by general infection, threatening life.

**Therapy.** Immediate isolation. Injection of Behring's serum beneath the skin of the abdomen or the eyelid. Frequent cleansing of the conjunctival sac with sublimate 1:5000 or potass. perm. 1:5000.

Membranes and coatings are to be removed only when this can be accomplished by a gentle brushing, and forcible divulsion of deep-lying membranes is to be avoided most carefully.

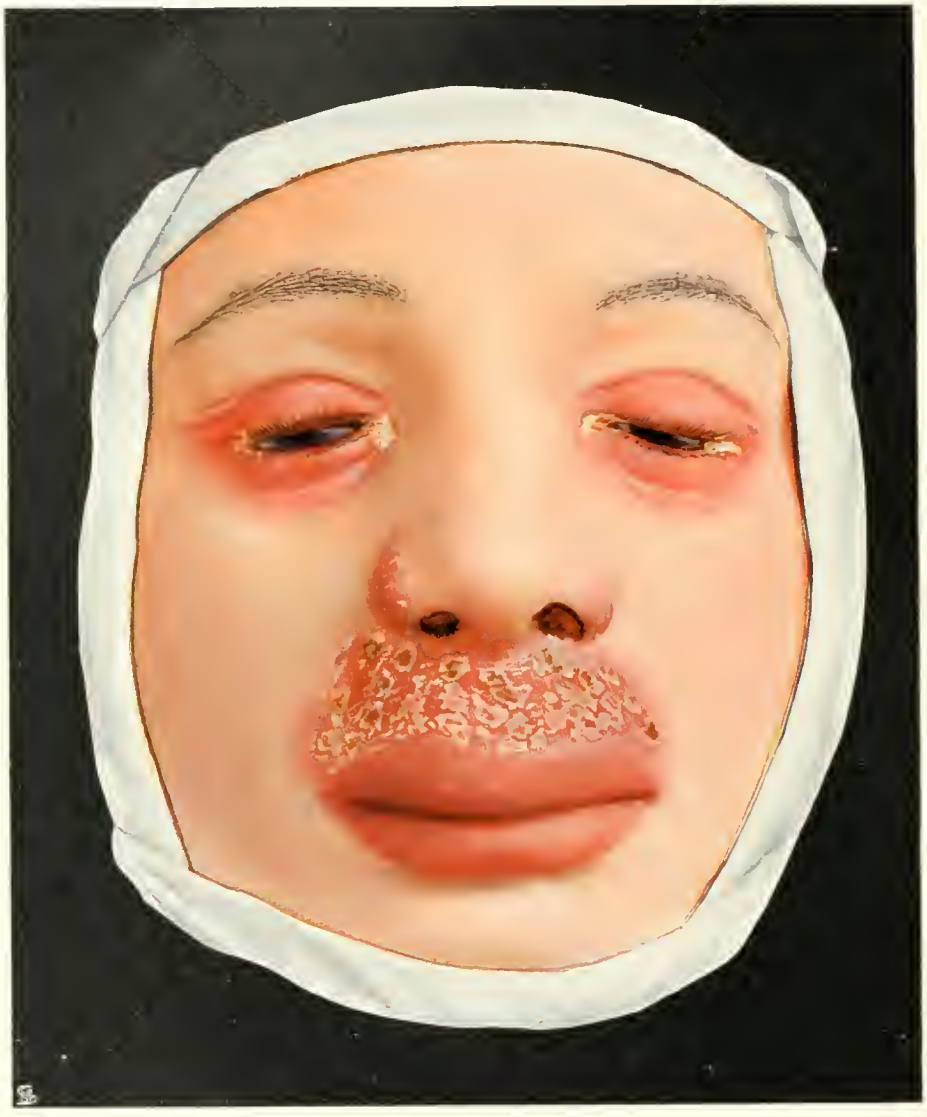

Fig. 41.
Blepharitis and Conjunctivitis eczematosa.

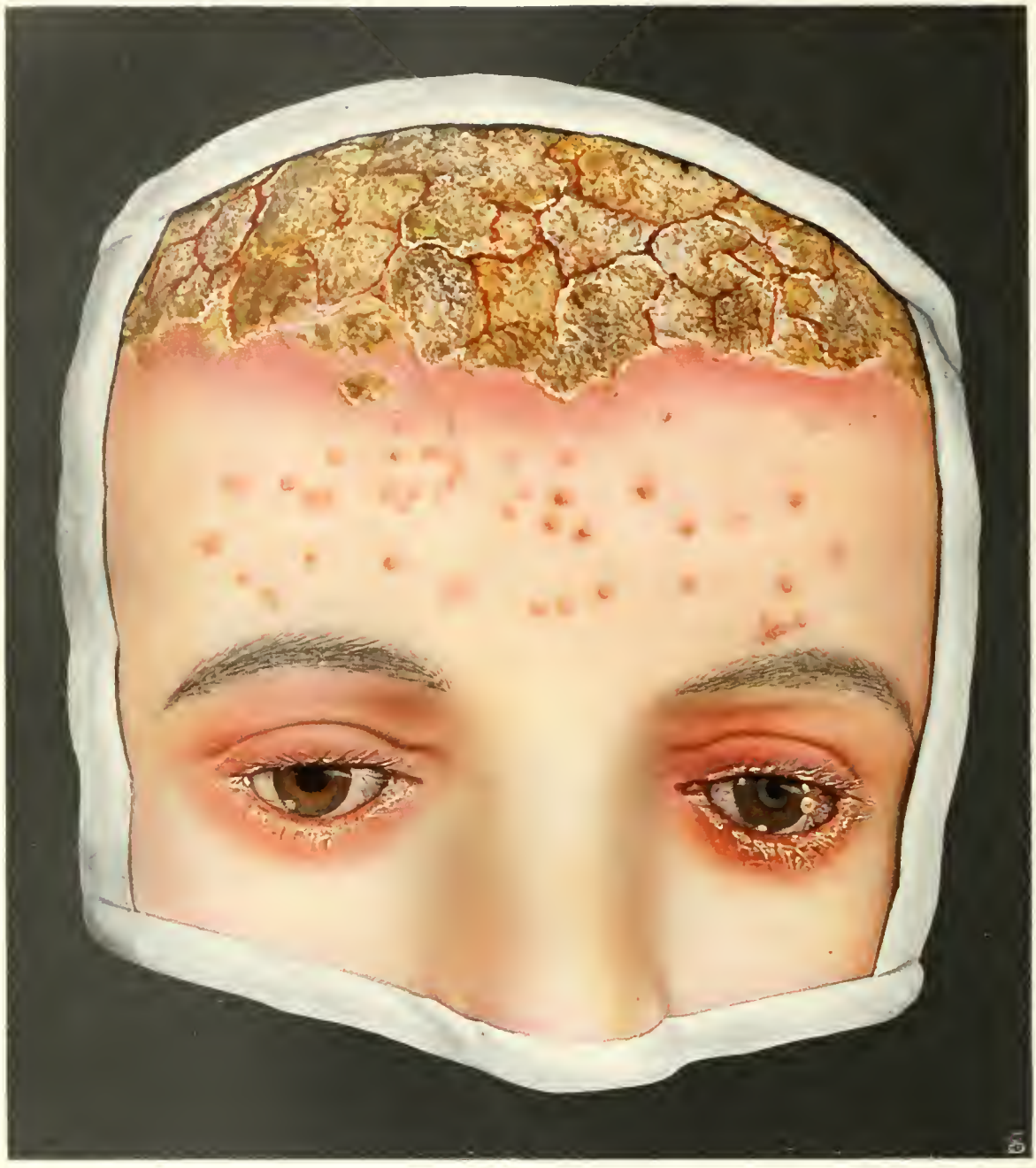

Fig. 42.
Conjunctivitis and Keratitis phlyctaenulosa.
Eczema faciei.

# Conjunctivitis and Keratitis Phlyctænulosa.

Plate XXVIII., Fig. 41; Plate XXIX., Fig. 42; Plate XXXII., Fig. 47.

The above disease boasts a numerous array of synonyms. Besides conjunctivitis phlyctænulosa we have C. eczematosa, C. scrofulosa, C. lymphatica and keratitis superficialis.

The disease is characterized by an eruption of phlyctenules, *i. e.* eczema pustules on the surface. These are cone-like elevations of the epithelium about the size of a millet seed, and their favorite location is the corneo-scleral boundary of the margin or limbus. where they are apt to develop in rows (*vide* Plate XXIX., Fig. 42). Soon a number of superficial blood-vessels are seen extending toward the growth, and these develop likewise in the superficies of the cornea after the appearance of phlyctenules in that region (Plate XXXII., Fig. 47), in which case the lesion is called: keratitis fasicularis.

After a somewhat brief period, the phlyctenules rupture, *i. e.* the apex of the cone falls off, and we have left small gray ulcers which heal rapidly and become covered with epithelium. The disease may pass through these phases within 3-4 days. Commonly, there is also present an eczema upon head or face. As a rule, the basic factor in the disease is scrofulosis.

**Prognosis.** The individual phlyctenule may run its course rapidly and safely, but relapses invariably

occur as long as the primary disease remains untreated. If the phlyctenules persist, they cause corneal infiltration which heals cicatricially, the scar being termed a macula corneæ.

**Therapy** must first take into consideration the underlying scrofulosis. Diet, iron, cold baths, brine treatment, living in the open air and all the hygienic measures suited to scrofulous children. Since obstipation often exists, the treatment should begin in children with the internal administration of calomel, after which the eczema of the head, nose or ears, or on the face should be handled. Otherwise, relapses occur (*vide* Therapy, page 40). Once daily, application of unguentum flavum (hydrarg. oxid via humid. parat 0.1 : vaselin. flav. 10.0) should be made to the area affected, spreading it over the surface by movements of the lids. In the later stages, the cicatricial cloudiness of the tissues may be cleared up by insufflation of the finest calomel powder.

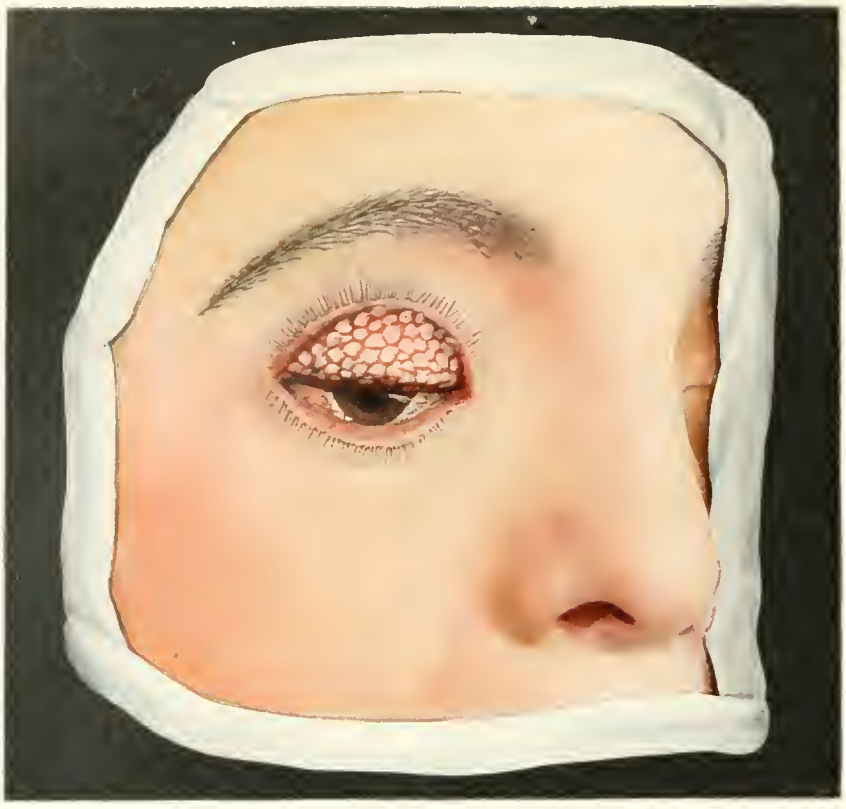

Fig. 43. Conjunctivitis vernalis.
Changes in the Conjunctiva.

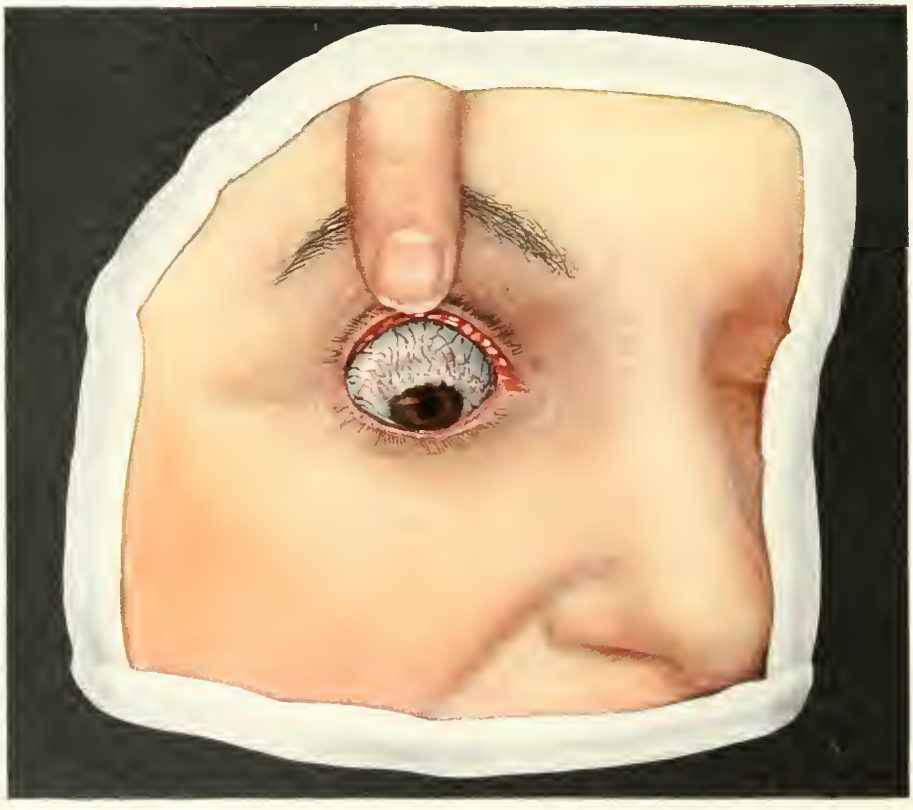

Fig. 44. Conjunctivitis vernalis. Changes in the Limbus.

# Conjunctivitis Vernalis.

Plate XXX., Fig. 43 and 44.

Conjunctivitis vernalis or spring catarrh is a somewhat rare disease, affecting chiefly the male sex in youth. The disease begins in spring-time, usually in both eyes, and after some months disappears or becomes milder. It continues thus, with relapses, for 3 or 4, even 10 and more years.

Those afflicted are greatly annoyed by itching and burning in the eyes.

There are two very characteristic anatomic changes in conjunctivitis vernalis; the first above the tarsus of the upper lid, the second at the limbus corneæ. Attacks may occur where only the one or the other change is present, and many authors therefore differentiate a palpebral form and a bulbar form. Usually, however, both conditions develop, and these are the characteristic cases. Others do not exhibit these typical anatomic insignia, but we find the conjunctiva injected and with an equally distributed thickening. In such cases diagnosis may be arrived at only by the history.

In palpebral tissue-changes, the conjunctiva tarsi exhibits broad but flat papillæ which, when numerous, give this part of the lid the appearance of being set with small cobblestones. These papillæ often have a diameter of several millimeters, an elevation somewhat less. They have a rounded periphery, unless pressed flat and angular by neighboring growths; the superior surface is flattened from above, or even some-

what concave. The papillæ feel hard to the touch. Horner fittingly compared the growths to colonies of mould. Sometimes they are of the density of cartilage. The conjunctiva above the excrescence and adjacent to it, has a peculiar, blue-white, milky shimmer, so that its surface appears pallid rather than inflamed. The "mushrooms" are soon present in large numbers, sometimes crowded together, sometimes isolated, thus giving the impression of having been rubbed off, their primary seat distinguished only by the delicate furrows left.

The conjunctiva of the lower lid is, as a rule, only thickened, without furrows, and of the same pallid, milky appearance, whilst tissue alterations of the fornices, violent inflammation and follicles are lacking.

The changes in the conjunctiva bulbi are much more conspicuous and earlier observed. Correspondent to the palpebral fissure, we note some of the peripheral blood-vessels approaching the limbus corneæ, and in the limbus itself are found hard, knobby elevations of a bluish-white tint, and found mostly situate on the internal and external margin of the cornea. They may also be seen as a narrow band of thickened, gelatinous consistency encompassing the entire limbus. Sometimes the elevations are flattened, and may persist for years; in other cases, nodular tumors are developed from them.

Anatomically, we have not a syndrome of conjunctival inflammation, but a purely hypertrophic process in the areas affected. The epithelium, in particulate, is much thickened, giving to the conjunctiva its macroscopic, bluish-milky luster. Superficially, the elevations are usually three times thicker than the norm, so that often there are as many as 30 cell strata superimposed, one upon the other. The epithelium also sends cell-cords into the deeper tissue, cells in cords and

nests with the formation of the most varied figures, the syndrome reminding one of carcinoma, but not penetrating so deeply into the tissues.

**Diagnosis.** The disease may be sometimes confused with trachoma, but the compactness of the elevations, their pave-stone appearance, the milky shimmer permit its recognition. The diagnosis becomes certain if we also have present the characteristic thickening of limbus tissue.

The **prognosis** is favorable in that the disease finishes its course without injury to the eye, but unfavorable since no remedy is capable of abbreviating the process.

**Therapy.** Strong cauterization of the affected areas is to be shunned for it only aggravates the condition. The use of a mildly astringent collyrium, as in conjunctivitis catarrhalis, is indicated here. A xeroform or anesthesin dusting-powder or the instillation of acid. acet. dil. 1:1000 several times daily is commended for the relief of itching. For the proliferations in the limbus, massage with 2 or 5% yellow precipitate unguent may be employed.

When the papillary proliferations become very large, they may be removed with scissors or the galvanic platinum loop.

# Tumors of the Corneo-Scleral Margin. Xeroderma Pigmentosum.

Plate XXXI., Fig. 45.

Epibulbar tumors usually develop in individuals of middle or advanced age, and begin almost invariably in the corneo-scleral margin, very seldom in the central area of the conjunctiva bulbi. According to Virchow, there occasionally appear deeply pigmented connective tissue cells in the corneal margin, also small black flecks (melanomata) composed of such cells, and these are genetic in the formation of new growths. Often no reason for their development can be discovered; in other instances they follow some slight injury or evolve from scar-tissue in the margin of the cornea. Long-continued irritation, such as working in a dust-laden atmosphere, is presumably favorable to their genesis. In most cases, a pigment fleck in the limbus conjunctivæ is first seen, which slowly attains a fungoid growth. Sarcomata usually have a smaller stem or trunk, extend more superficially, are of soft consistency, have many blood-vessels, and look like more or less pigmented, reddish nodules. Carcinomata develop a wider growth. At first glance, they often appear as if firmly and extensively attached to the corneal surface, but a careful examination with a blunt sound demonstrates that it is a mere superimposition and that, generally, there is no union of tissues.

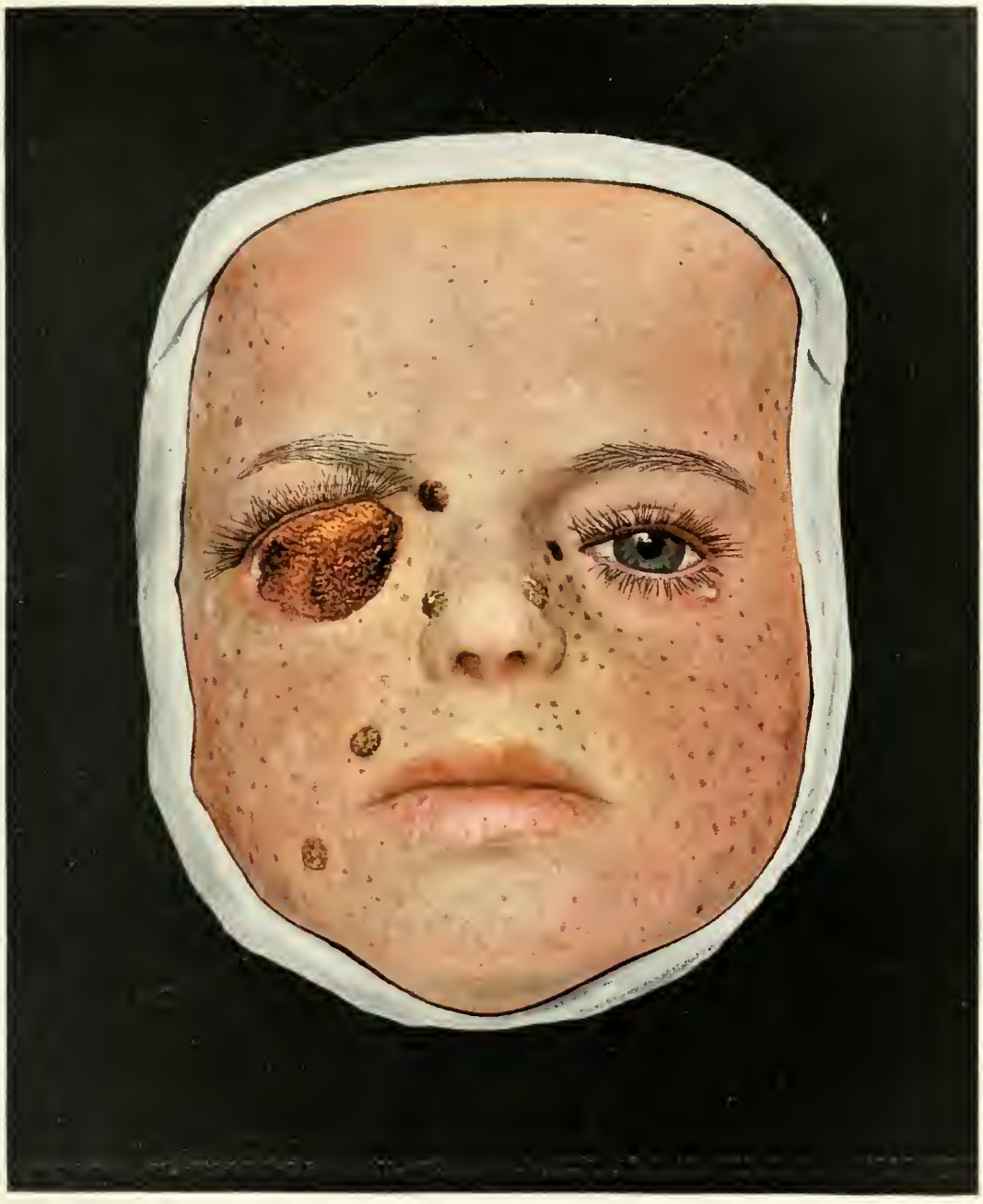

Fig. 45.
Xeroderma pigmentosum. Tumor epibulbaris.

Progressing backwards, they unite with the conjunctiva and with it are movable over the eyeball, a sign that the neoplasm has not penetrated deeply.

If the tumor be not removed, its periphery slowly extends, attacks conjunctiva and cornea, and in time forms an enormous neoplasm.

If patients come, as is commonly the case, with more or less pigmented, fungoid tumors of the size of a lentil or pea, the thorough removal of the neoplastic tissue suffices. The nodule is severed with scalpel or scissors, and its base cleaned up with the sharp curette, followed by cauterization with the Paquelin. A soft tumor is generally rooted in the superficial layers of the tissue. One should not fail to warn the patient of the malignancy of the growth and emphasize the necessity of his appearing from time to time in the consultation room.

In a number of cases, the removal of the tumor leads to a cure, yet relapses occur in a majority of cases, though these exhibit an extraordinarily slow growth. With very large and extensive tumors, the exenteratio orbitæ or enucleation of the eye is, conditionally, commended, though in such cases there has occurred metastasis to the inner organs, and the patient dies from exhaustion or tumor formation elsewhere.

### Xeroderma.

By xeroderma pigmentosum we mean a peculiar morbid syndrome first described by Kaposi (1870) in his textbook on Diseases of the Skin. Altogether, up to the present day there have been reported a little over 100 cases, which is not many when we consider that the growths are sometimes multiple, most apt to be among members of the same family, and further that, with the great interest aroused by a case, it is unlikely that a single one has escaped publication.

Some cases have also been reported several times by different authors.

The disease is accepted by most authors as due to a congenital pre-disposition, to which corroboration is given by the fact that the morbidity in almost all of the reported cases was found in children of the same family, *e. g.* in one instance in seven brothers. And, in single cases it affected only children of the same sex, in other cases both sexes were involved, facts similar to those observed in other inherited diseases. In our own cases, there were two brothers affected, in the elder of which the disease was far advanced. But, neither in our cases nor in those of others were there any morbid conditions in the parents demonstrable as having any definite relationship with the disease affecting the children. Neither consanguinity, constitutional or dermal conditions, nor even weakliness were present. The children were born with a normal derm, but even in the first period of extrauterine life, the initial phenomena appeared. Under the action of the sun's rays during the first, or, at most, the second year of life, there developed on the exposed portions of the body, face, neck, hands and forearms, on the feet and legs of children going bare-foot, circumscribed, red spots which disappeared in a short time with slight desquamation, but invariably reappeared after further exposure to the sun. Lukasiewicz himself was able to observe a diffuse redness of the skin as the initial stage of the disease. As often as his little patient remained but a short time in the open air on sunny days, there appeared after a few hours a diffuse reddening and swelling of the diseased areas of skin. The rubescence paled under pressure of the fingers, which caused pain. There was no elevation of temperature but a well-marked state of depression in the patient. For a few days these symptoms were

aggravated, then diminishing with slight degeneration. There was frequent repetition of the above syndrome, particularly in spring and summer, whilst in winter, when the patient remained indoors, the erythema never made its appearance.

In typical cases, permanent alterations soon occur. During this period, which may be termed the second stadium, pigmentation sets in. On the exposed parts of the body, numerous freckle-like spots develop, whilst elsewhere pigmentation diminishes, so that there are areas free of pigment and perfectly white. Altogether, the pigmentation increases so that the affected areas, compared to the normal skin, appear brown and even black.

Further along in the disease, there is much vascular dilatation, usually in the form of small, flat but numerous telangiectases, more rarely angiomatoid tumors.

The skin, in general, becomes atrophic, smooth, and the normal furrows and folds disappear. If a piece of such skin be examined, there is found a degenerative process analogous to that of senility in the sense of an atrophy, a thinning and flattening of the papillæ and their epiderm.

With this, we find, microscopically, a typical penetration of the rete cells into the chronically altered cutis, hyperplasia of the sebaceous glands and ectasia of individual blood-vessels (Lukasiewicz). These furnish the needful conditions for the development of the last stage of the disease: multiple carcinoma. Here and there, correspondent usually to folds of the skin, elevations appear which develop into wart-like processes so that often the brown-black areas of the affected parts are wholly covered with them. From a greater or less number of such nodules, there then develop genuine epithelial carcinomata, which, though like

other epithelial cancers, are steadily progressive, by disintegration lead to enormous ulceration, or by reason of the gradually increasing cachexia cause death, though apparently without metastasis to internal organs.

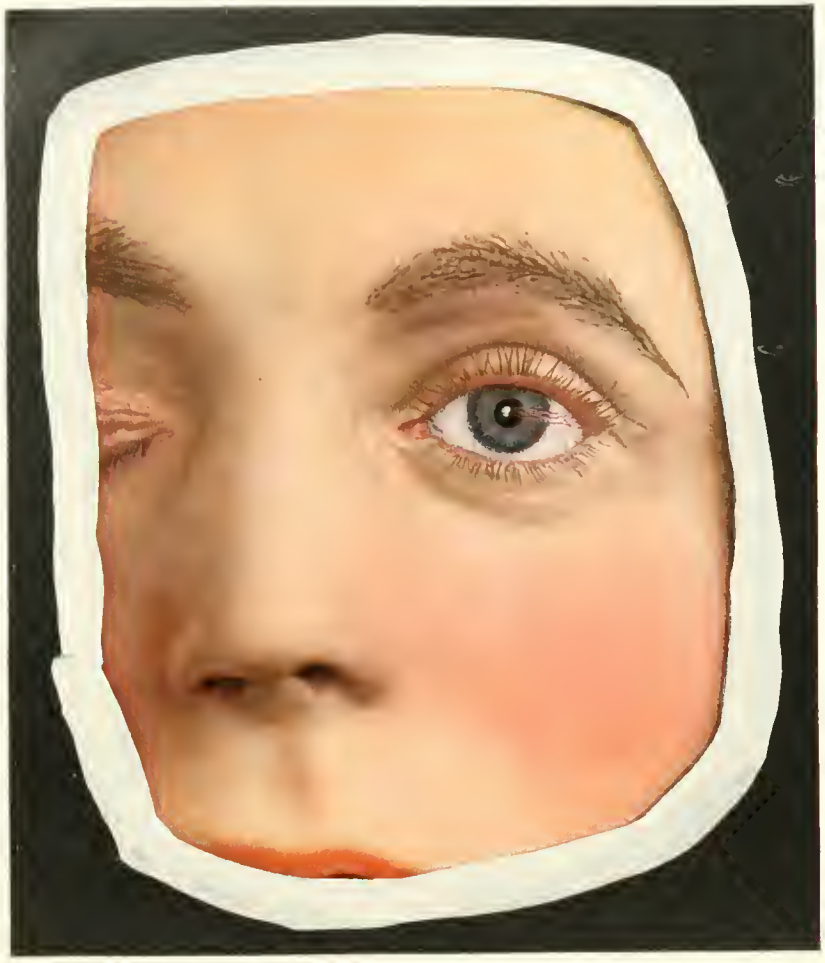

Fig. 47.
Keratitis fascicularis.

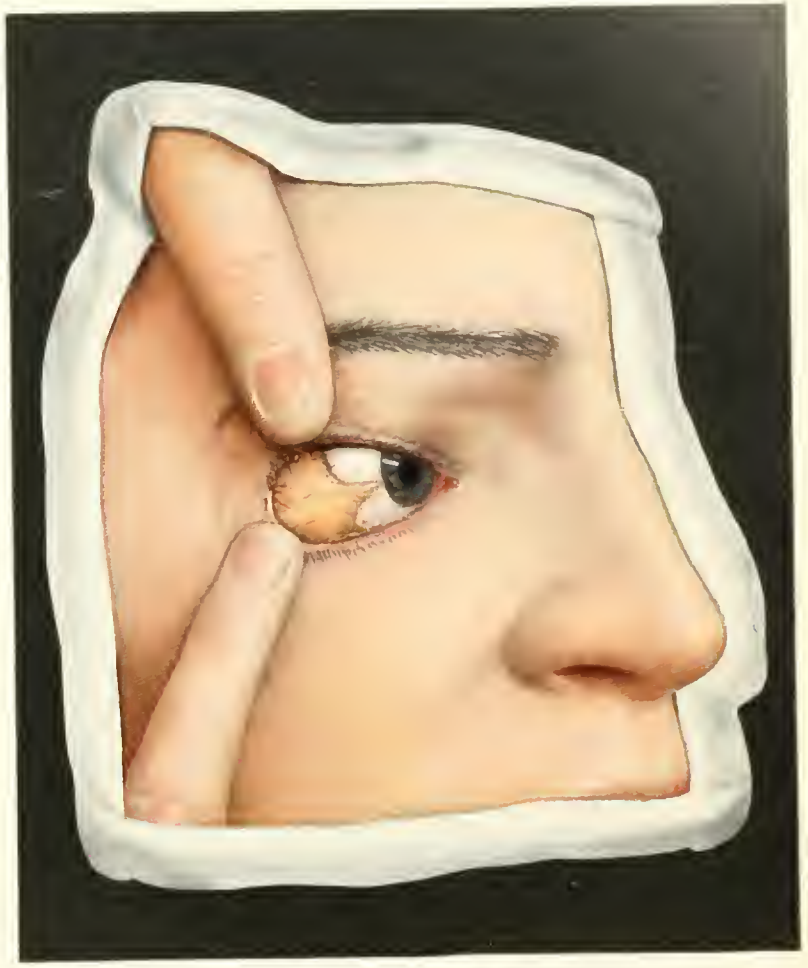

Fig. 46.
Lipoma subconjunctivale congenitum

# Lipoma Subconjunctivale Congenitum.

Plate XXXII., Fig. 46.

This lipoma is a not very infrequent congenital tumor, located in the external canthus beneath the conjunctiva bulbi and between the insertions of musculus rect., externus and the musculus rect. superior. With the conjunctiva (somewhat thickened over the tumor), it is easily movable upon the subjacent tissue. The lipoma, commonly the size of a lentil or pea, has a yellowish shimmer through the overlying conjunctiva. Toward the corneal side the tumor appears shorn off, cliff-like, whilst on the temporal side it gradually flattens and passes into the fatty orbital tissue. If the lipoma be small, it may not be noticed at first glance, and is first seen by a side-glance toward the nose. Microscopically, it is composed essentially of hyperplastic fatty tissue.

**Diagnosis** of this lipoma is infallible, for no other tumors are found in this region.

The **prognosis** is absolutely favorable. The tumor may begin its growth at puberty, but is always comparatively benign, though it may push out through the palpebral fissure, and thus hinder exact approximation.

**Therapy.** If small, the tumor may be left to itself. It is better, however, to remove it, as it may cause disfigurement. The extirpation of the fatty mass after splitting the conjunctiva is simple and need not be absolutely radical.

# Keratitis Parenchymatosa Sive Interstitialis.

Plate XXXIII., Fig. 48; Plate XXXIV., Fig. 49-50; Plate XXXV., Fig. 51; Plate XXXVI., Fig. 52.

(Synonyms: Keratitis interstitialis, profunda, diffusa, syphilitica, uveitis anterior.) Parenchymatous inflammation of the cornea is a distinctly characterized disease, usually beginning and running its course in a very typical manner. Exact knowledge of it is the more important because it must always be considered as a constitutional malady. Its appearance, therefore, renders it the duty of the physician to make an accurate and thorough examination of the patient, for, with the commencing keratitis parenchymatosa as the first symptom, there will almost invariably be found other symptoms pointing to a hitherto latent constitutional disease, in most cases syphilis hereditaria (keratitis syphilitica or keratitis ex lue congenita).

As for the name given the disease, keratitis parenchymatosa is most used, though not the best, for, of course, all diseases of the cornea implicate more or less its parenchyma. Keratitis syphilitica correlates it with the etiology, but is not suited to all cases, and, moreover, is not characteristic of direct lues, but only of inherited syphilis. Keratitis profunda denotes correctly that the morbid process is carried on in the deeper layers, but corneal ulcers also penetrate the deepest strata. Hence, keratitis interstitialis or keratitis diffusa would serve best for the equable, diffuse

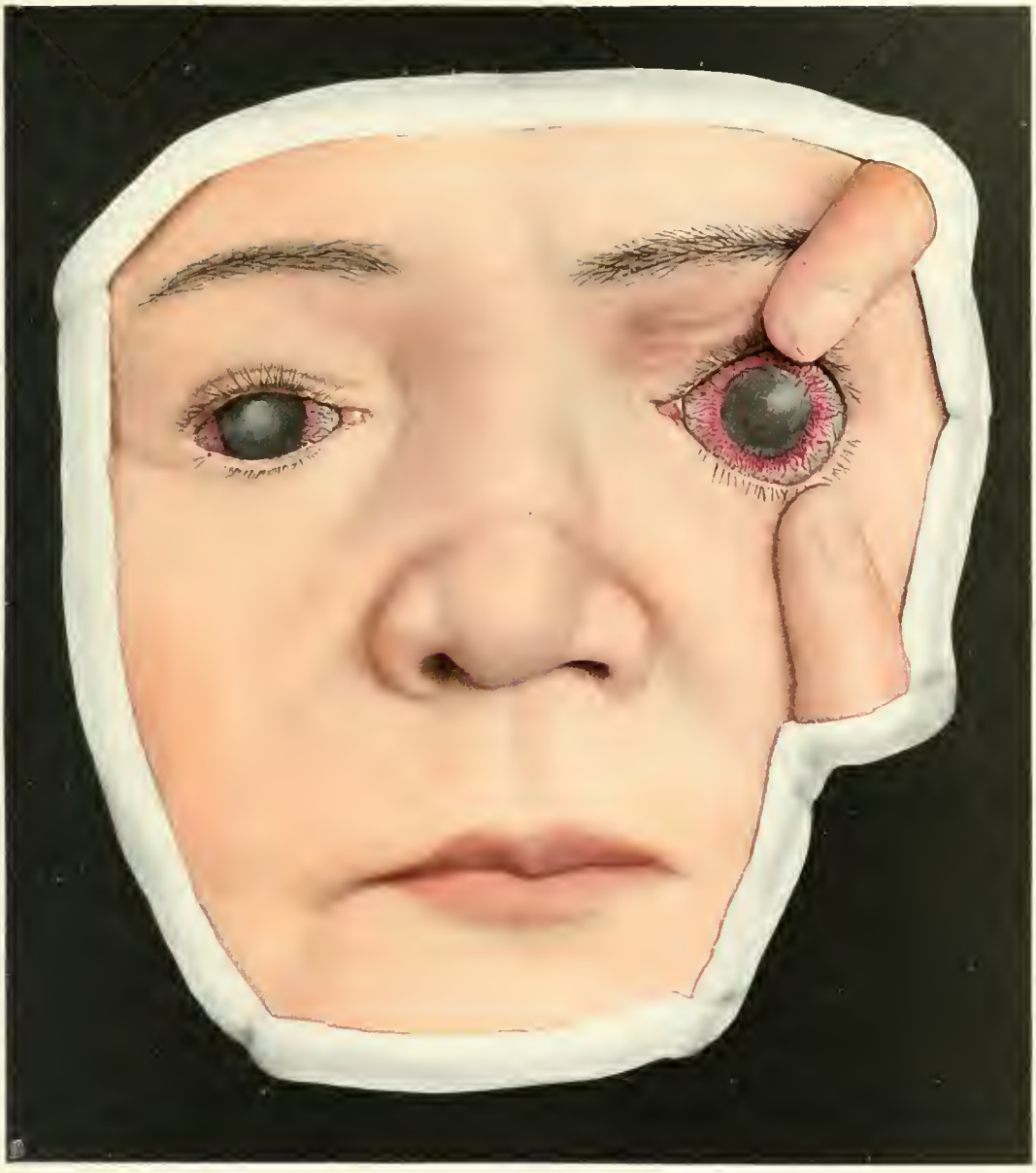

Fig. 48.
Keratitis interstitialis.

progress of the disease over the entire cornea, which is perfectly characteristic. The recently justified term, uveitis anterior, we shall discuss later.

The disease usually begins in one eye, though also in both, with a delicate pericorneal injection, soon augmenting in intensity and immediately pointing to a more violent and deep ophthalmic affection. Soon there is noticed in some part of the cornea a turbid, lusterless area, generally triangular in form, beginning at the periphery and thence extending to the center of the cornea.

The turbidity, observed with the naked eye, seems uniformly gray, but with a lens or even by focal illumination it is seen to be composed of a number of small, whitish flecks. To this turbid area first discovered, others are added which become confluent, and finally implicate the entire cornea in the same manner. The cornea looks like a piece of glass which has been breathed upon or rubbed over with fat, and the deeper-lying parts but shimmer through it or else are barely discernible. In differentiation from superficial keratitis it is important to note that the corneal surface exhibits no gross changes, no nodules, vesicles, etc., nor indentations, epithelial defects or ulcers. But with the lens we see that the epithelium is raised up in many minute elevations equally distributed over the whole corneal surface, giving it a finely granular appearance. This is beautifully seen with the keratoscope of Placido; nowhere are the concentric rings, mirrored against the cornea, broken, but the circumferences exhibit rounded margins or edges.

With the development of corneal turbidity, there begins an extensive vascular proliferation in its tissue. The new-formed vessels extending from the marginal network stop at the limbus or pass just into it, forming a reddish plaque around the cornea. The deeper-

lying episcleral vessels, on the contrary, frequently push into the deep corneal strata, their advance always being a little behind that of the corneal opacity, with whose progress, however, they advance further. In mild cases, few blood vessels are found in the cornea, and these are often imbedded in the opacity and difficultly seen. When the malady is more violent, the vascular growth is steadily forwards until the entire cornea is finally of a dirty, raw-meat hue. These blood-vessels have a very characteristic appearance. Whilst in pannus trachomatosus they usually twist and interlace, here they pursue a direct course in parallel; even after dividing, the branches run directly and in parallel toward the center, thus resembling the hairs of a brush.

Simultaneously with the parenchymatous keratitis, there develops a more or less noticeable involvement of the uveal tract, varying from a simple hyperemia of the iris to an intense irido-chorioiditis. The frequent, almost invariable iritis is betrayed by the pericorneal injection, the swollen, discolored appearance of the iris, and the tendency of the pupils to contract. If, in this stadium, there be no artificial dilatation of the iridic opening, intense cases will develop a permanent posterior synechia and even occlusion of the pupil. Even after the frequent instillation of atropin, the pupil often exhibits the tendency to contract. If one has the opportunity to use the ophthalmoscope at the beginning of the disease whilst the cornea is yet transparent, he will see in the anterior portions of the fundus oculi, large, black spots, indicating a chorioiditis or uveitis anterior. This chorioiditis would probably be considered one of the most frequent phenomena accompanying keratitis parenchymatosa, did not the opacity of the cornea rapidly interfere with ophthalmoscopic examination.

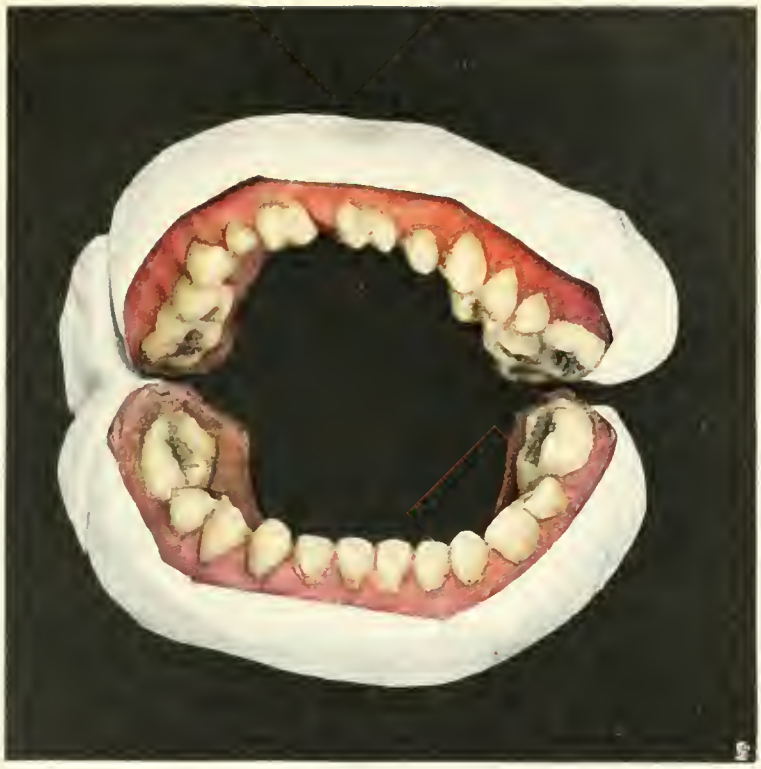

Fig. 49. Hutchinson's Teeth.

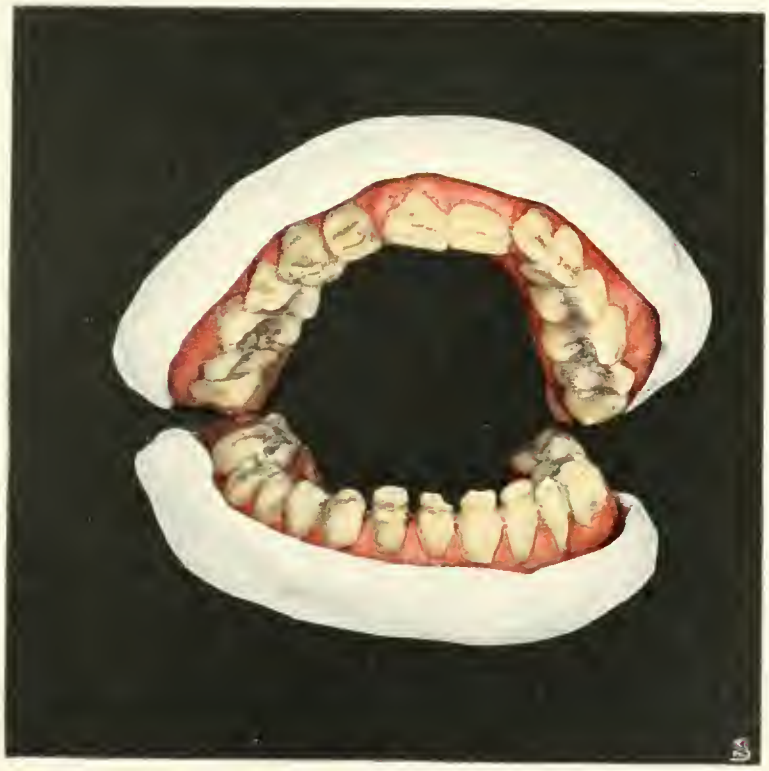

Fig. 50.
Rhachitic Teeth.

In some cases, alterations of intraocular pressure occur, it being occasionally lowered. Increase in pressure is rare, and develops only after long continuance of the disease.

Vision in this affection is always considerably lessened. Generally, the coarsest print cannot be read and only motions of the hand are cognized. Both the testing of vision and the examination of the eye are hindered by the often violent photophobia and the accompanying profuse lacrimation of the affected eyes. In the initial stage of the disease, there are often strikingly few subjective symptoms and no pain, but later there is sufficient pain, though if the disease be properly handled, the pains are not intense. Photophobia, as a rule, troubles the patient most.

To the disease syndrome, with its complications, above described, may be added, according to the intensity of the inflammation, a number of variations, but the disease commonly presents enough of the characteristic symptoms to make diagnosis easy. As contrasted with a superficial keratitis, one should note that here we have no ulceration and almost never any gross elevations of the corneal surface, so that in keratoscopic observation, though the contours of the circles be not sharply defined, they are still concentric and circular. Furthermore, the general habitus of the patient aids in the determination of an extremely important differential diagnosis. Superficial keratitis is, usually, a symptom of the scrofulous constitution with its nasal troubles, eczemas, glandular affections; parenchymatous keratitis is commonly a symptom of hereditary lues.

Keratitis parenchymatosa almost always attacks both eyes, not always synchronously but with a short interval of time between; more rarely there are intervals of weeks and months. The disease usually de-

velops between the 6th and 18th years of life. Exceptionally, individuals over 20 years of age have been attacked, and these cases, as a rule, run an atypic and milder course, and with them in rare instances, one eye alone may be affected. The disease commonly appears in pallid, unhealthy children who appear badly nourished.

**Etiology.** The disease is never a local affection. Formerly, it was classified as keratitis lymphatica seu scrophulosa and Hutchinson was the first to declare it due, as a rule, to hereditary lues. At the same time, he called attention to another symptom often found with it and considered as a certain indication of existent hereditary syphilis, *viz.* Hutchinson's teeth (*vide* Plate XXXIV., Fig. 49).

By Hutchinson's teeth we mean a dental form where the two middle upper incisors of the second dentition have, instead of a straight cutting-edge, a half-moon notch or indentation and converging sides. This notching of the free margin of the tooth is commonly observable up to the 25th year, after which the corners are broken or worn off. With this typical form are often found other dental abnormalities, *e. g.* abnormal smallness of the canines, wide intervals between teeth and irregular location and formation of the teeth in general.

Such teeth are not to be confused with rachitic teeth, in which we find horizontal furrows and ridges and defective enamel (*vide* Plate XXXIV., Fig. 50).

With the two symptoms described, the interstitial keratitis and the Hutchinson's teeth, there is most frequently associated a third, *viz.* hard hearing, and this, as a rule, without objective clinical cause. The three phenomena form the so-called Hutchinson triad, and they diagnose absolutely the existence of hereditary

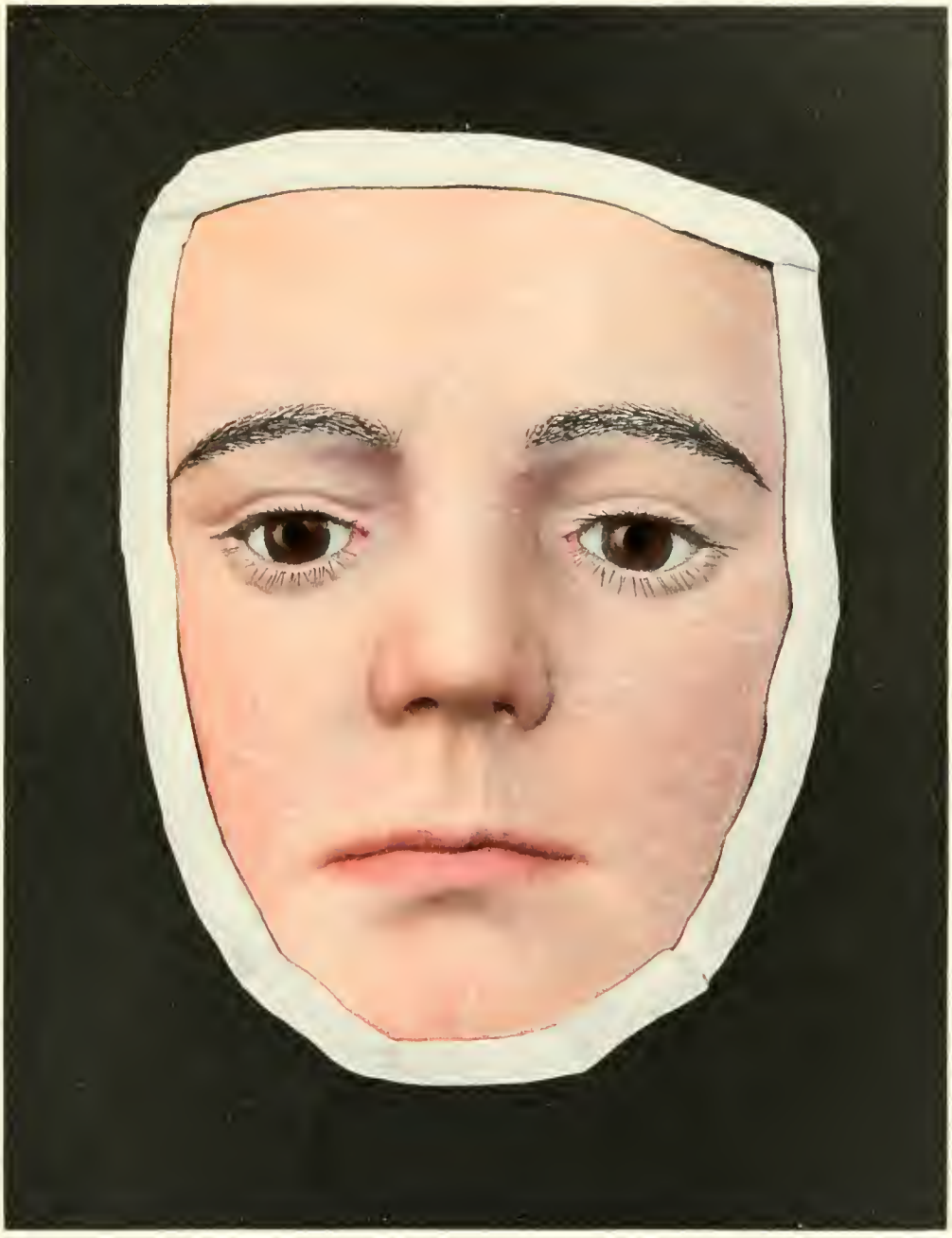

Fig. 51.
Rhagades on face in hereditary Lues.

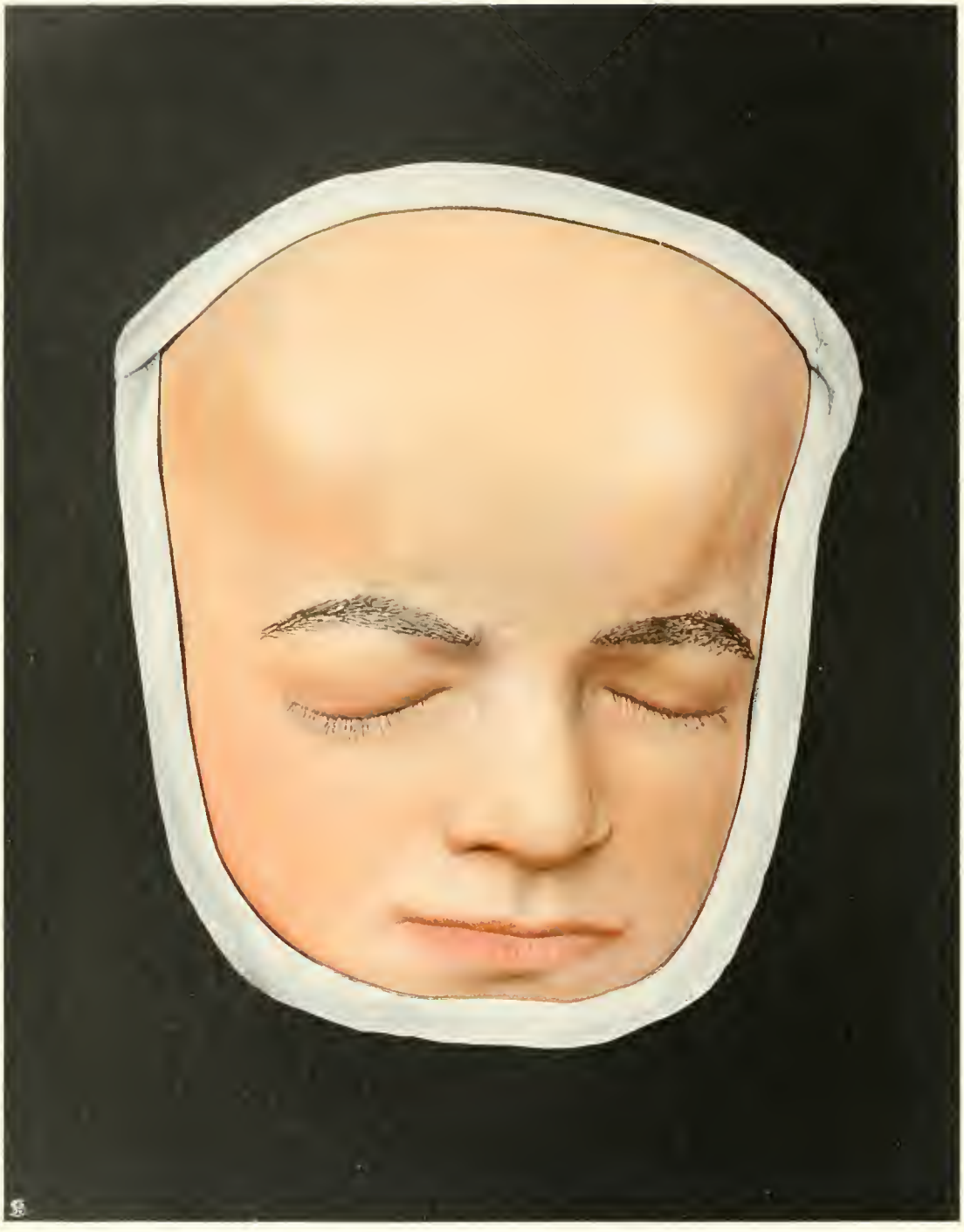

Fig. 52.
Head formation in hereditary Lues.

syphilis. I have, however, seen no case where the Hutchinson triad was present and with it, other symptoms.

Arlt and Förster have called attention to a frequently occurring affection of the knee-joint, either preceding or following the disease.

Recently, more attention has been given to the frequent occurrence of articular troubles in hereditary syphilis. According to Fournier, these were found 82 times in 212 cases.

The knee is most often affected, then the elbow, more rarely, other joints. The articular inflammation generally precedes or develops with the interstitial keratitis, seldom following it. The articular trouble often develops on both sides of the body.

We are concerned here almost invariably with serous effusions into the joints, usually sequent to moderate, drawing pains and beginning without fever or with but slight elevation of temperature. According to Fournier these articular troubles are dependent upon affections of the bones, whilst other syphilographs hold that they may be primary synovites.

We should also note the state of the lymph glands, the peculiar formation of face and skull (*vide* Fig. 52, Plate XXXVI.), the often sunken nasal bridge (Fig. 48), an ozena perhaps present, blenorrhea of the lacrimal sac, rhagades at the corners of the mouth or over the entire face (*vide* Plate XXXV., Fig. 51), etc.

Together with the objective findings, the anamnesis is important. Inquiry should be made concerning a possible earlier infection of the parents, whether premature or dead children preceded the birth of the patient, whether many children died in infancy, etc.

Often a glance or two will establish the diagnosis; often the most careful investigation and examination are necessary, and there will always be cases

where hereditary lues cannot be considered. In such cases, other diseases are etiologic, such as scrofula, chlorosis, and above all, tuberculosis. Of rarer etiologic factors, we may mention articular rheumatism, and finally, malaria and influenza.

**Course.** The disease runs a very slow course, its briefest duration being several months and it is often six months or a year before the inflammatory phenomena subside and the opacities clear up. As an average we may reckon on a half year. It is therefore recommended that the physician inform the patient of the possible duration of the disease and admonish him to cultivate fortitude. And likewise, when the disease begins in one eye, the patient should be told of its rapid appearance in the other, lest he be terrified at the new outbreak, and also that he may not become suspicious of the therapy employed.

The **prognosis,** despite the fact that no medical assistance can abbreviate or arrest the disease, is relatively favorable. Even after long duration, the corneal opacities usually clear up, the process commonly beginning at the margin so that the center of the cornea is the last to clarify. As a rule, the eye regains a fair or, at least, endurable degree of vision, and rarely are there any dense opacities left.

**Therapy** must be local and constitutional. In the beginning, warm, moist compresses commonly mitigate the symptoms of irritation and inflammation. The compress is made by placing a large, moist piece of absorbent cotton over the eye, then some guttapercha tissue followed by a gauze roller to keep the compress in place. Renewal of the dressing once or twice daily may be indicated. Another method of attaining the same result is frequent irrigation or

bathing of the eye with warm water. The warm, moist compress is particularly serviceable at night when a permanent bandage is required. The best solution with which to moisten the cotton is a 2-4% boric acid, the ancient household friend, chamomile tea is less cleanly, and apt to carry dust particles into the eye. As soon as iridic involvement is noted—and this is seldom absent—the first and most important measure is the repeated instillation of atropin (1%) until the pupil dilates fully, a procedure which must be again practiced later in the disease. If these measures be neglected, posterior synechia, possibly complete occlusion of the pupil will surely ensue, and the eye will be permanently and seriously injured. Furthermore, the eye should be well protected against the light by moderate darkening of the room, and later, by a sun umbrella and protective eyeglasses.

When there has been a long-continued, excessive vascular development, a peritomy of the cornea has been done, *i. e.* with a scalpel a circular corneal incision through the blood-vessels has been made or else a circular strip, 1-2 mm. wide, removed at the corneoscleral junction. Experience, however, has taught us that the blood-vessels unite again after a time, and a more violent state of irritation has so often followed the operation that most ophthalmologists have abandoned it. If the inflammation have subsided, we endeavor to clear up the opacity by irritant measures, such as: the insufflation of calomel powder or massaging with yellow ointment, which is used at the Berlin University Clinic in somewhat less strength than that originally prescribed by Pagenstecher.

As already emphasized, it is extremely important to note the general constitution of the patient. In feeble individuals a roborant diet is indicated (good food, eggs, meat, etc.) and iodine preparations are partic-

ularly desirable (iodide of iron, cod liver oil with iodine, iodine mineral waters). In the later stages, living in the country, in good air, is to be strongly recommended.

In the numerous cases where hereditary syphilis is etiologic, this, naturally, should receive first consideration, using the ordinary remedies, mercury and iodide of potash.

If tuberculosis be the specific cause or fundament of keratitis parenchymatosa, the nutrition of the patient should be first attended to. Internally, kreosote or its active principle, guaiacol (which is less unpleasant to the taste and better borne) is recommended.

Malaria, of course, demands the administration of quinine, and, in the various forms of rheumatism, treatment by sweating, together with sod. salicyl., aspirin, or light-baths.

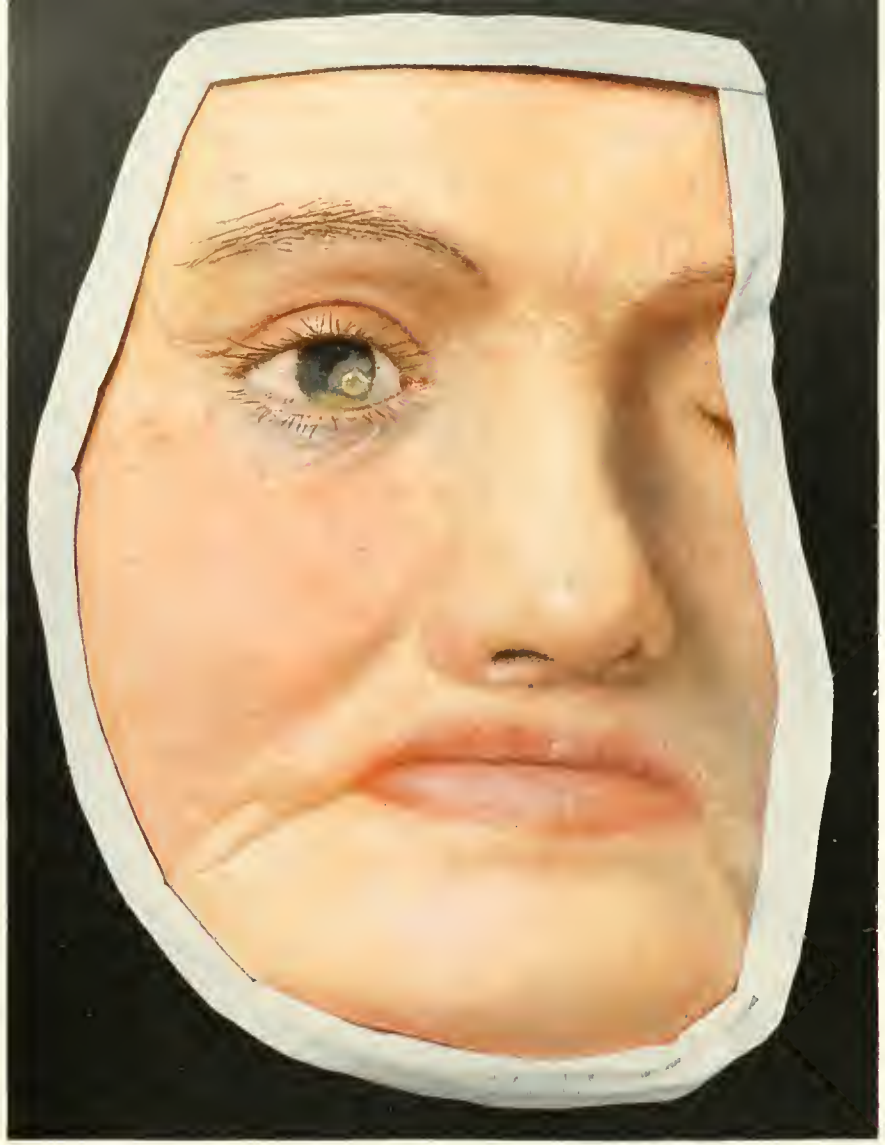

Fig. 53.
Ulcus serpens with Hypopyon.

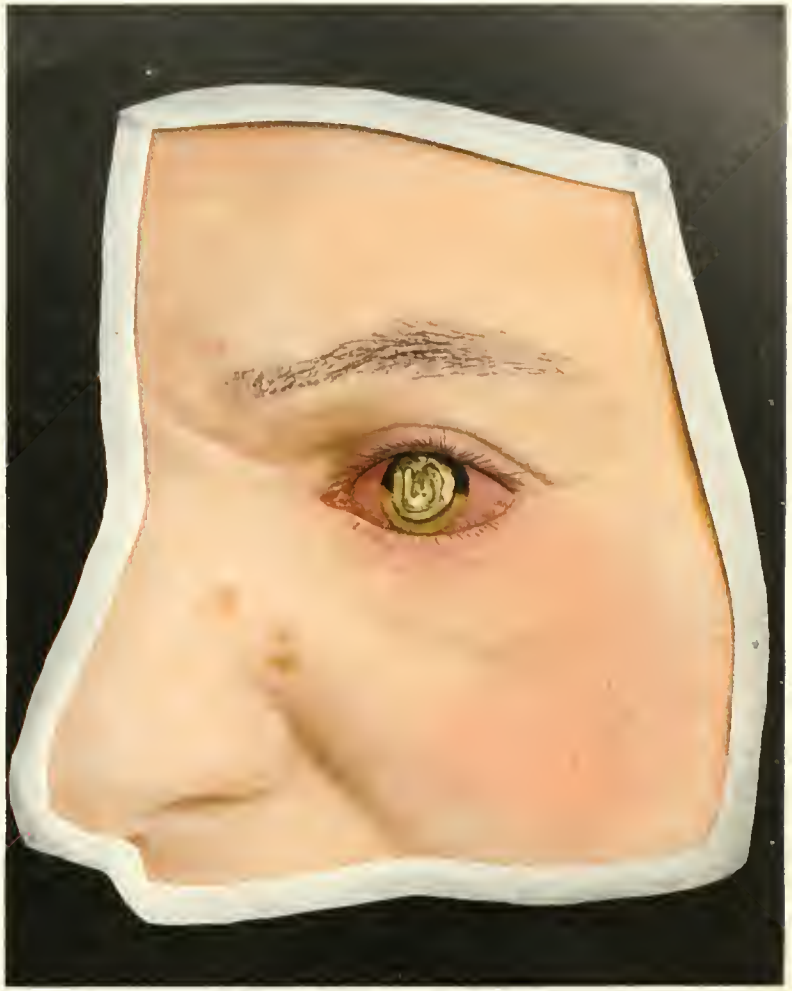

Fig. 54.
Advanced Ulcus serpens.

# Ulcus Serpens Sive Hypopyon-Keratitis.

Plate XXXVII., Figs. 53-54.

The normal epithelium of the cornea is so dense in structure, that the common excitants of suppuration are unable to gain entrance. Pus containing staphylo- or streptococci cannot penetrate the intact cornea, and even the virulent pus found in a dilated lacrimal sac and in which pneumococci are chiefly found, may constantly and for a long time flow over the eye without causing any noticeable inflammation, the cornea, at least, remaining unaffected. Gonococcal pus alone is able to penetrate intact corneal epithelium, though this takes considerable time, and is due to maceration of the epithelium by stagnating pus. Other excitants of suppuration require an artificial entrance for their penetration into corneal tissue, and such entrance is usually due to a wound, even if only a slight and superficial abrasion. If pyogenic germs present in the conjunctival sac, on or in foreign bodies, in the secretions of the lacrimal sac then enter the wound, the colonies which flourish luxuriantly beneath the epithelium cause its desquamation and there develops what modern science most suitably terms a septic ulceration of the cornea. It is characteristic of this lesion that there soon develops in the anterior chamber a collection of pus, called a hypopyon (whence the older nomenclature, hypopyon-keratitis). Because of its pronounced tendency to burrow rapidly and uninterruptibly be-

neneath the epithelium, the name, ulcus serpens, is very descriptive.

As soon as the ulcer has attained a certain growth, iritis regularly sets in.

In Fig. 53 is seen the disk-shaped ulcer with its yellow, up-turned margin. The circumjacent cornea is edematously opaque and misty. Below, in the anterior chamber lies the narrow, sickle-shaped hypopyon, and a commencing pericorneal injection denotes the initiation of an iritis.

Fig. 54 shows an ulcus serpens which has destroyed the greater part of the cornea. The hypopyon fills more than half of the anterior chamber. There is a slight palpebral swelling, and a marked pericorneal injection denotes the existence of an intense iritis which we are no longer able to observe directly.

**Prognosis.** Ulcus serpens, a frequent and serious disease of the eye, will, if left to itself, lead within a few days to permanent blindness. It is, therefore, a grave condition and the cause of much blindness. After eating off the entire surface of the cornea, the process commonly works downward until a rupture occurs, after which it usually halts. The loss of substance caused by it, is replaced with scar-tissue.

**Therapy.** In many cases the source of infection is in some diseased condition of the lacrimal sac, which should be split open from the outside and tamponned with iodoform gauze or else thoroughly extirpated, for the use of the sound is profitless. Every two hours a few drops of undiluted and freshly prepared aqua chlori should be poured upon the ulcer, when, if it still advances, the platinum loop should be used in cauterizing the edges. Application of iodoform powder follows the cauterization, or better, airol in the form of a fine powder placed within the con-

junctival sac where it forms a paste, soon hardening to a firm crust over the ulceration. In far-advanced cases the cornea should be obliquely split (method of Sämisch).

A pneumococcic serum has been manufactured lately and used subcutaneously with success in ulcus serpens.

# Leucoma Corneæ from Variola Vera.

Plate XXXVIII., Fig. 55.

Only the epithelium of the cornea regenerates and becomes transparent again. After destruction of corneal substance, there develops an opaque cicatrix of various degrees of density and thickness, according to the thickness of the lost substance. A delicate, superficial, yet visibly gray scar is called a nubecula; a thicker one, macula; a perfectly opaque, white scar, a leucoma corneæ.

This last form is either partial, as in Fig. 35, Plate XXIV., or total, as in Fig. 55.

In many leucomas, a piece of the iris has become adherent, forming a synechia anterior or leucoma adhaerens (seen in Fig. 35 above the black, streak-like scar), a sign that perforation of the cornea has taken place.

**Therapy.** In thick leucomas there is nothing to be hoped for from therapeutics or from time. If they cover the entire pupil, vision is lost save for a sense of light. If partial, an artificial pupil may be inserted in the remaining portion of the cornea (*vide* Fig. 35). To lessen the disfigurement, India ink has been stippled into the leucoma, thus simulating more the black iridic aperture.

In slight corneal opacity, a certain degree of cure is got through time alone. This may be accelerated by irritants (insufflation of calomel, pencilling over with

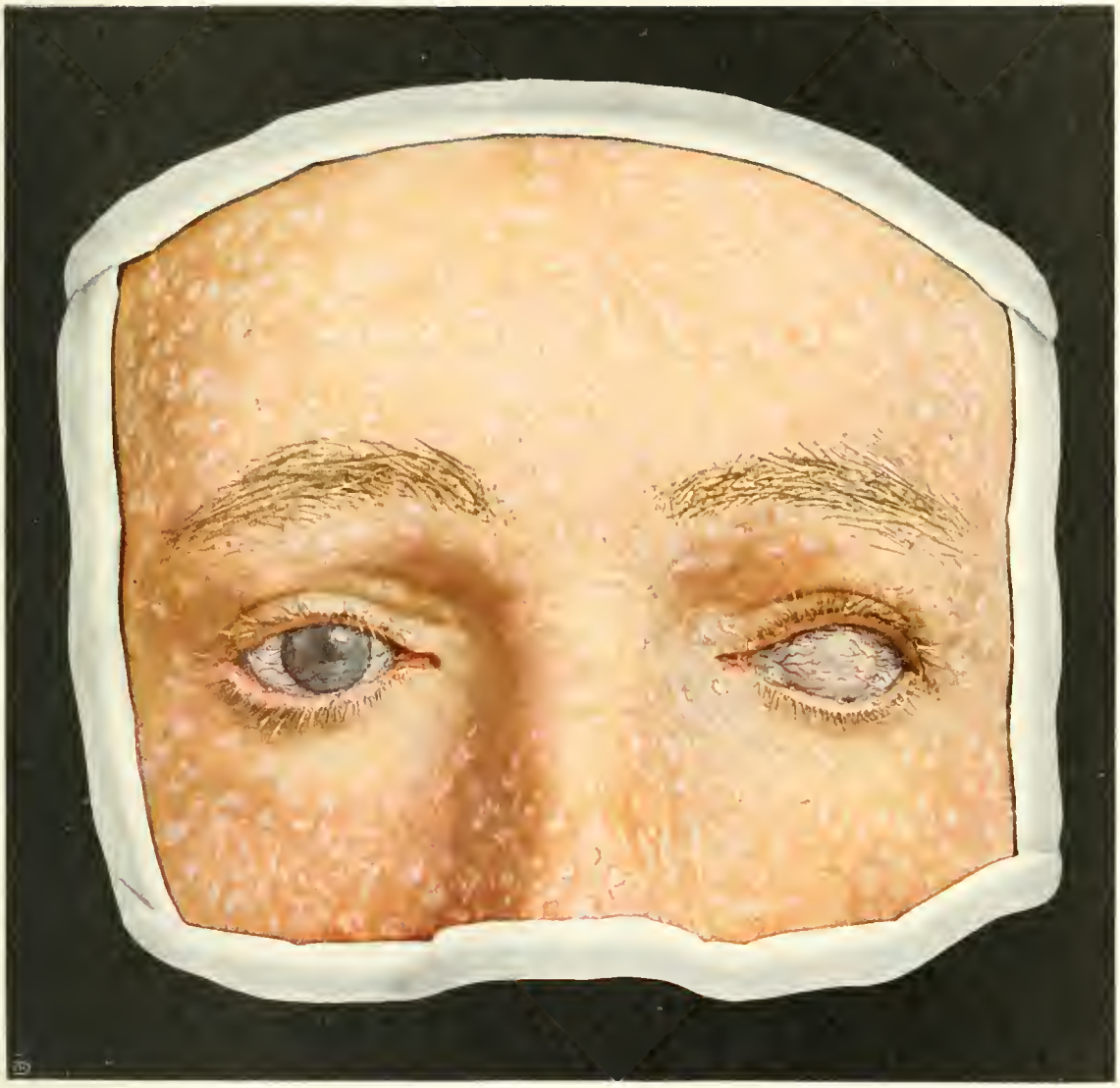

Fig. 55
Leucoma corneae totale.

yellow mercuric oxide salve, 2-5%, and by hot vapors), to induce more rapid metabolism, but, such therapy is suitable only in fresh cases. Recently dionin (5-10% sol., later in substance or powder form) has been much recommended.

There are, also, congenital opacities; the corneæ of both eyes have a milky and sclerotic appearance. These opacities are not defects of development, as formerly believed, but the residua of a keratitis interstitialis in utero e lue hereditaria. Treatment, then, must be antisyphilitic.

A non-inflammatory, physiologic opacity is the arcus senilis or gerontoxon corneæ, appearing, sooner or later, with advancing age. The opacity is characteristic—a narrow, gray line, concentric with the corneal margin. It almost invariably begins at a point in the superior margin; then the lower margin develops a similar bow or arch. With the further extension, the superior and inferior sections meet at the inner and outer sides, and the ring or circle of the arcus senilis is complete. Its outer side is sharply marked and invariably separated from the limbus by a strip of transparent cornea (*vide* Plate L., Fig. 74).

**Prognosis.** The arcus senilis is not a progressive opacity, for when the circle is completed and has attained a breadth of 1-2 mm. the process stops.

To the non-inflammatory corneal turbidities belongs the girdle-like or ribbon-shaped opacity (*vide* Plate XXV., Fig. 36). It is a broad, white band (2-4 mm. in breadth) extending horizontally just below the middle of the cornea. A narrow, transparent line of normal corneal tissue separates the opaque band at its two extremities from the corneal margin. It develops slowly in the course of years and usually in eyes blinded by an insidious irido-chorioiditis. It is due to a

deposition of lime salts following a disturbance of nutrition in the cornea.

**Therapy.** There is none. As it generally develops in eyes already blind, it is a condition of no practical significance.

Fig. 56.
Keratoconus.

Fig. 57.
Staphyloma corneae partiale.

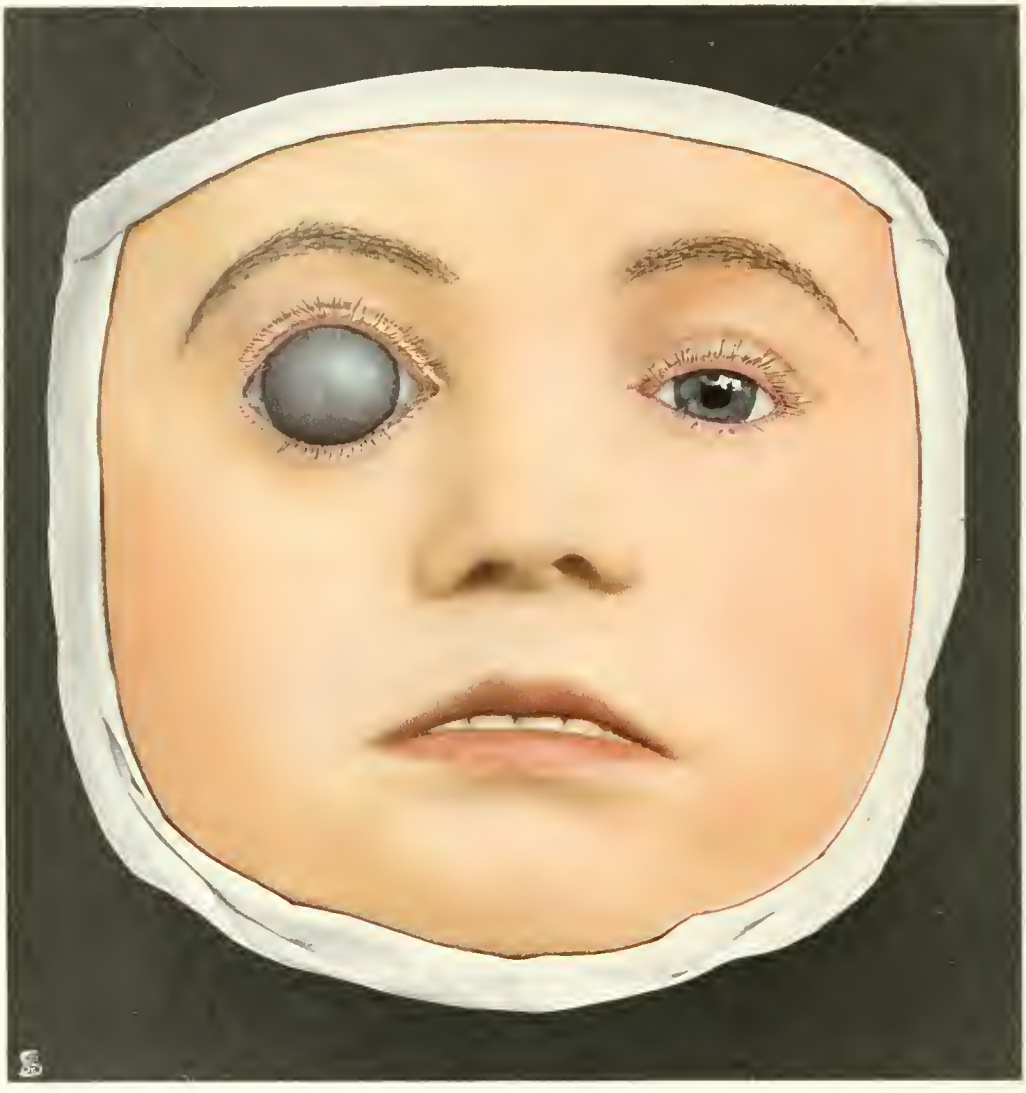

Fig. 58.
Buphthalmus. Cornea globosa

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# Ectasias of the Corneæ.

Plate XXXIX., Fig. 56: Keratoconus.
Plate XXXIX., Fig. 57: Staphyloma corneæ partiale.
Plate XL., Fig. 58: Cornea globosa; Buphthalmia.
Plate XLI., Fig. 59: Staphyloma corneæ totale.

(a) Among corneal ectasias, staphyloma corneæ should be first mentioned. It is composed of a prolapsed, cicatrised iris, and, therefore, at its point of exit, the cornea has been entirely destroyed, and the intraocular pressure forces forward the yielding cicatricial tissue of the iris. We differentiate a staphyloma partiale (Fig. 57) and totale (Fig. 59). At first, and with small staphylomas, the black pigment of the iris is predominant in the extruding mass, but later the iris becomes so cicatrized and enlarged that the staphyloma appears gray-white, traversed by dilated blood-vessels.

Staphyloma is the end-product of a corneal suppuration. Its genesis is shown in the left eye of Fig. 38, Plate XXVI., where we see how, after destruction of the cornea, there has developed a total prolapse of the iris, which has already begun to buckle forwards.

**Therapy.** A partial staphyloma must be removed. The open wound then closes usually with firmer connective tissue than the delicate iridic tissues are capable of producing.

After removal of a total staphyloma, the resulting large wound is best sutured together, which, however, leaves but the stump of an eye.

(b) Keratoconus is a rare disease generally affecting both eyes. Very gradually and with no inflammatory phenomena, the central portion of the cornea begins to protrude forwards, cone-like. This corneal area remains transparent for a long time, until finally the apex of the cone, subjected as it is to enormous dilatation, begins to develop opacity from cicatrization.

The etiology of the disease is unknown. Because of some disturbance of nutrition the central portion of the cornea becomes thinner and hence so much the less able to withstand the anteriorly directed intraocular pressure.

**Therapy** is rather impotent. In conjunction with a strengthening diet, the instillation of a miotic (eserin, pilocarpin) may be practiced for a long period to reduce permanently the intraocular pressure, though an iridectomy for the same purpose is better The apex of the cone may be destroyed by galvanic cautery in order to obtain a more resistant cicatrix.

(c) Keratoglobus-Buphthalmus. In keratoglobus or cornea globosa, the transparent cornea in all its parts is symmetrically thrust forward, so that instead of its normal "watch-glass" projection from the globus, its form resembles that of a "cover-glass" used to protect cheese. It is only one of the phenomena accompanying the general enlargement of the eyeball in hydrophthalmus or buphthalmus or the total sclerectasia resulting from increase of pressure in the eye of childhood, whilst the sclera is still yielding in its structure (in the first years of life). We have, therefore, an infantile glaucoma.

The nature of the disease, however, is not fully understood. Possibly it is due to a congenital misplacement of the natural exits for ocular fluids.

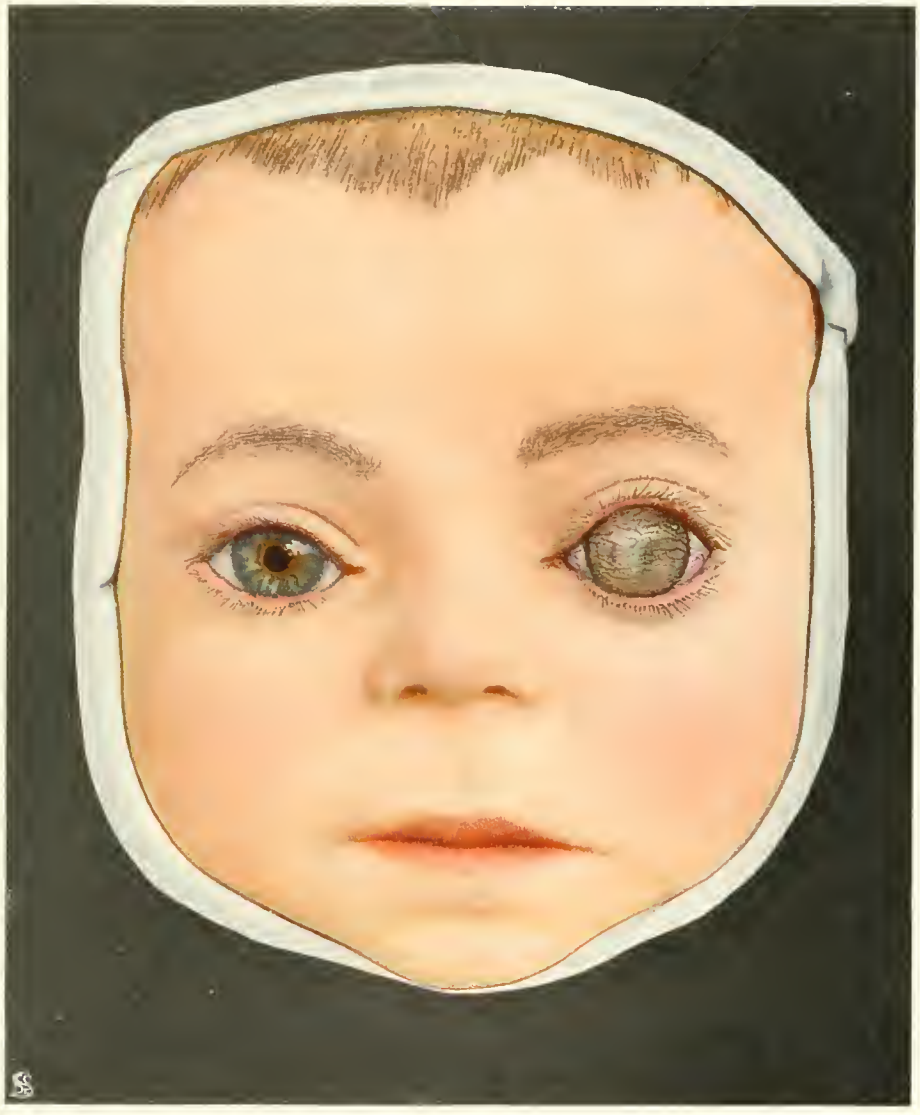

Fig. 59.
Staphyloma corneae totale.

The eye is often enormously enlarged in its periphery, and the attenuated sclera is bluish because of the perlucence of the chorioid. Anteriorly, the enlarged cornea, of a misty or milky-white opacity, stands out like a cheese cover glass against the bulbus. The anterior chamber is, likewise, very deep.

**Prognosis** is most unfavorable, and, left alone, the process gradually leads to absolute blindness, very rarely halting in its progress. Anatomically, there is a total glaucomatous excavation of pupil and globe.

**Therapy.** It is very advisable, to perform, as soon as possible, a broad iridectomy or sclerectomy.

# Lepra.

Plate XLII., Fig. 60; Plate XLIII., Fig. 61.

Lyder Borthen emphasizes as an important phenomenon, that the outbreak of the nodular form of lepra almost always begins in the eyebrows. It is diagnostically valuable to know that the loss of the eyebrows is the first, and sometimes for years, the only sign of the disease. As initial symptom in the eyebrows, the formation of nodules is more frequent than diffuse infiltration. The eyelashes also are regularly involved. The disease consists of the falling out of the hair, or atrophy, partial or total.

The skin of the eyelids is frequently and early diseased, either infiltrated or nodulated. The infiltration, often edematous in appearance, may be synchronous with that of the eyebrows, or, separated from it by healthy skin, may occur along the margins of the lids.

In the lids, the nodes themselves are particularly large, and most often located along the free palpebral margin. It is peculiar that the lid-nodules may appear symmetrically on the correspondent eyelids.

The maculo-anesthetic type, *i. e.* blotches or spots, is also often found in the eyebrow though not so frequently as in nodular leprosy.

If, in the nodular type, an infiltrated area of the skin of the eyebrow be excised and the humor pressed out, the diagnosis may be rendered certain by demonstration of the lepra bacillus.

The disease seems to begin in the middle layers of the skin, and usually in the center of the node a blood-

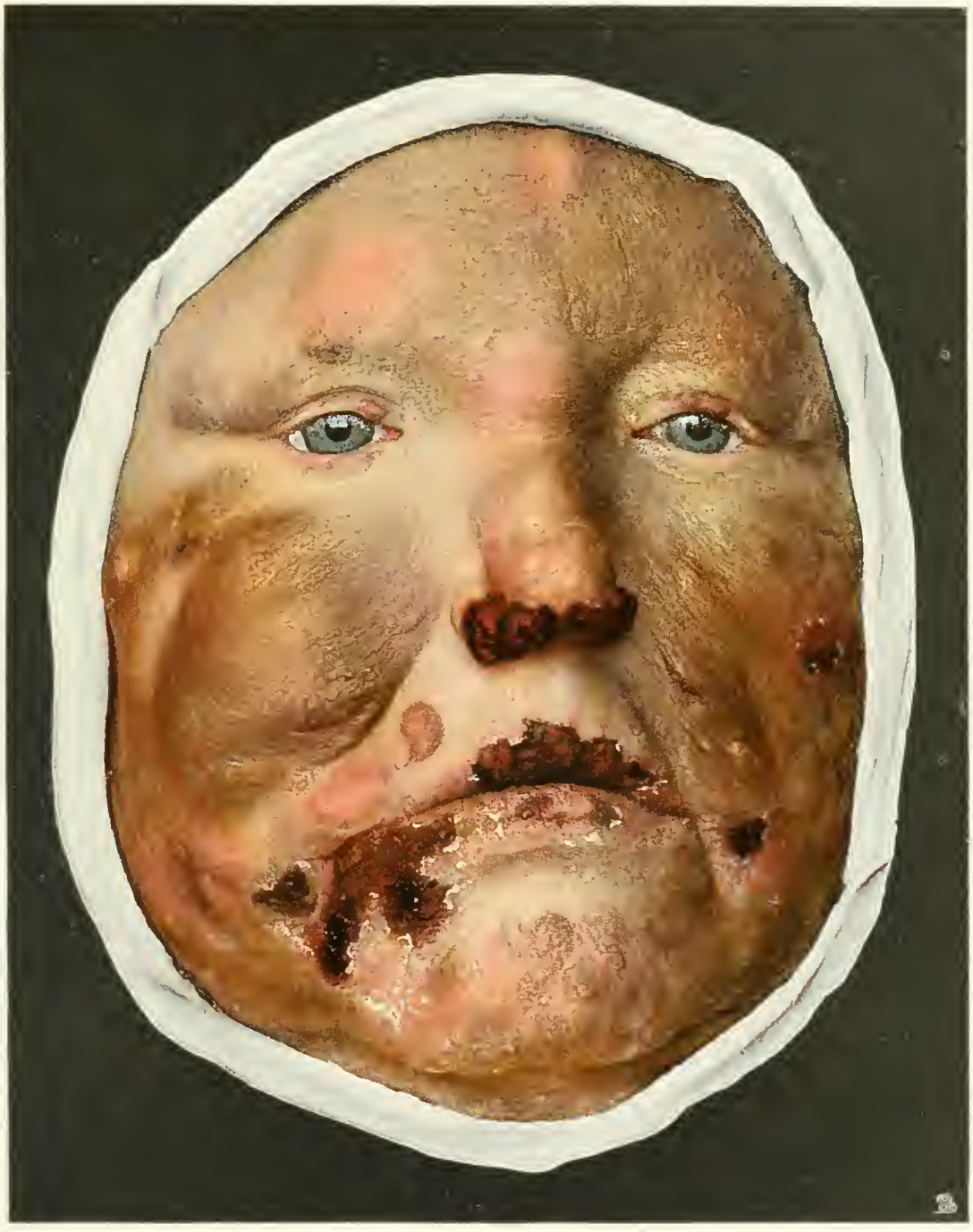

Fig. 60.
Lepra. Keratitis punctata.

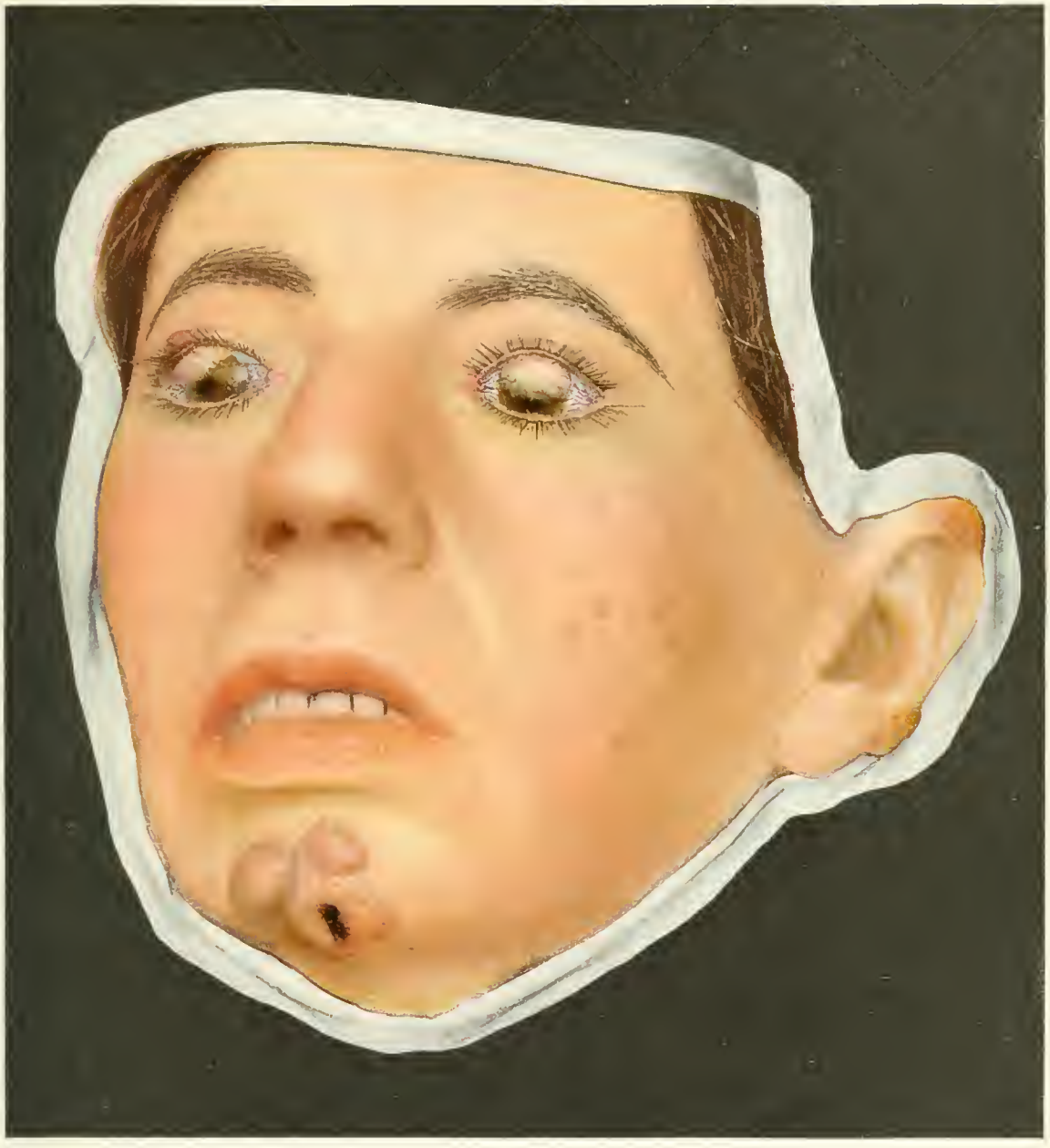

Fig. 61.
Lepra. — Epipulbar Leprome.

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vessel, lymph-channel, often a capillary, is demonstrable. The bacilli are, therefore, carried by both the blood and lymph systems. Not rarely they are found in the cells of the intima, seldom in the white corpuscles in the lumen of the blood-vessel. If capillaries or small vessels are the points of nodule formation, marked dilatation with hemic engorgement occurs with the bacillary invasion. Infiltration of the tissues surrounding the vessels develops late, and only when the bacilli begin to flourish outside of the vessel. Then there occurs an accumulation of emigrated leucocytes and a great increase of nuclei through the proliferation of the fixed cells of the connective tissue.

Further growth of the node is accomplished by the bacilli attacking the cells adjacent to the blood or lymph channel. The leprous virus is by no means as active as, for example, that of the tubercle bacillus, which, in a comparatively short time, kills the cells. Hence, in lepra nodes many bacilli can be seen in cells of normal appearance and still retaining their power of proliferation. This minimal tendency to destruction of cells permits leprous proliferations to take on the appearance of veritable tumors (H. P. Lie).

Affections of the eyeball are frequent, in fact, almost the rule, in lepra, and the most common is a keratitis punctata or nodosa, in which small white or gray nodules develop beneath the epithelium with very little ophthalmic irritation (*vide* Fig. 60). The nodules are chiefly composed of lepra bacilli.

A true keratitis parenchymatosa with iritis is not infrequent.

Finally, the granulation-tumors described as on the lids are also found as epibulbar upon the cornea, and, as with other epibulbar tumors they extend outwards from the limbus corneæ.

# Scleritis. Sclerectasia.

Plate XLIV., Fig. 62; Plate XLV., Fig. 63.

In inflammation of the sclera we may differentiate forms affecting only the superficial layers (episcleritis) from a true scleritis, confined to the middle and deep strata.

In the superficial form there is usually first found a circumscribed morbid focus close to the cornea. The area is puffed up, and forms a more or less steep hillock or hump, at first dark-red and later more of a blue or violet hue. It is hard to the touch, not movable, and very sensitive. Toward it and through it run dark-red, deep-lying (episcleral) blood-vessels, and the injected conjunctiva covering it is movable. The ocular surface elsewhere is almost non-irritable (*vide* Fig. 62).

This infiltration shows little tendency to destruction of tissue or to ulceration, and sooner or later the exudate is resorbed, though commonly with the destruction of some scleral tissue, for, in the area of inflammation, the sclera has a delicate smoke-gray tint which, with a higher degree of intensity, becomes slate-gray. We have to do, then, with an attenuation of the sclera (cicatrized tissue) through which the black chorioid shimmers, but, however, much less noticeable in this superficial form than in the deeper. The bulbus, furthermore, is commonly not injured.

The deep form of scleritis is rarer, and at first more difficult to diagnose, for it is not possible, without autopsy or dissection, to determine how deep the scle-

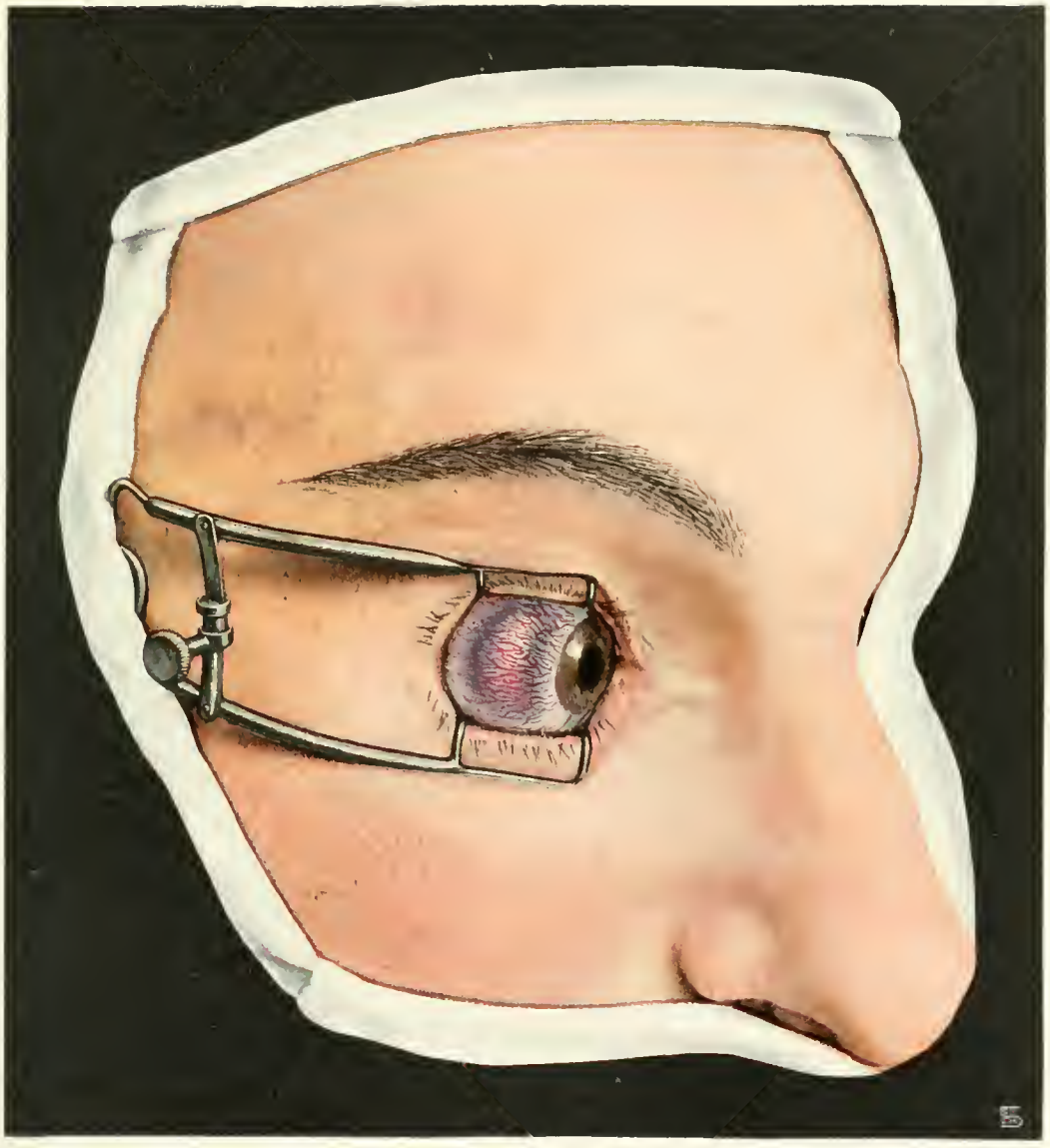

Fig. 62.
Scleritis.

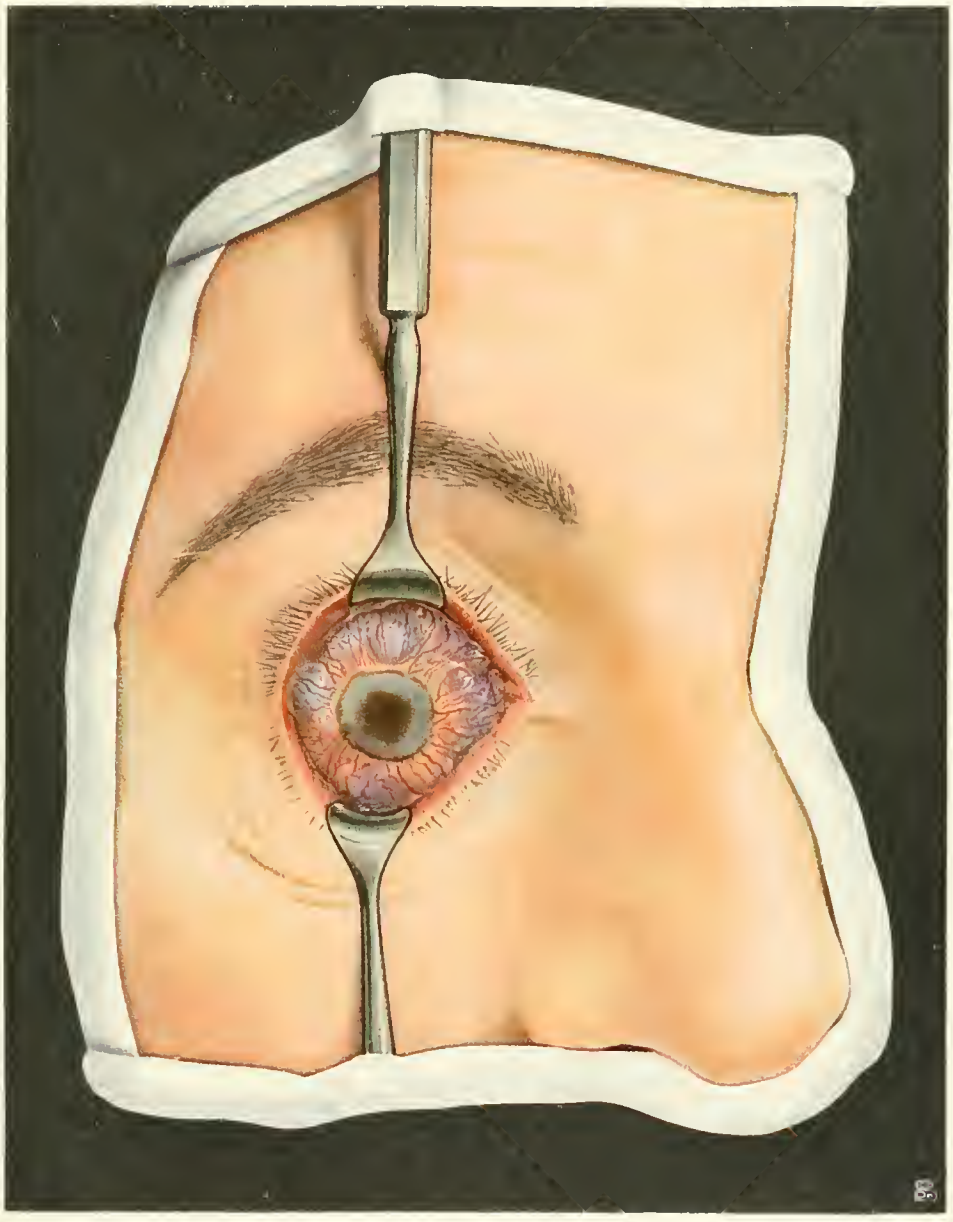

Fig. 63.
Sclerectasia.

rotic inflammation extends. We deduce this from the unfailing involvement of other ocular membranes (iritis, chorioiditis, sclerosing keratitis), and hence the disease is called scleritis complicata by many authors, and develops during its course the resultant marked thinning of the sclera, whose rupture is due to intraocular pressure. At the beginning we again find a dark-red or bluish hump-like projection or swelling in the affected area. The projection, in general, is not sharply circumscribed and has a great tendency to extend farther, so that later, the entire cornea is surrounded by these elevations.

In this form, also, there occurs no direct disintegration, and there is left, finally, only a marked, cicatricial thinning of the sclera, often so prominent that the chorioid beneath shimmers through with a bluish tint. Often, however, after resorption of the exudate, the sclera has lost so much of its firmness that it is unable to offer sufficient resistance to the intraocular pressure, and there results a bulging out,—ectasia,—of the affected areas.

Fig. 63 shows such anterior scleral staphylomata, which, because they often surround the cornea like a ring, are termed annular staphylomata. In such cases, however, the staphylomata do not become entirely confluent, but between the individual lumps there are always retractions, so that the comparison with a piece of large intestine or black-pudding sausage (Blutwurst) is not inapplicable.

The eye in Fig. 63 is held open by the Desmarre speculum; in Fig. 62 by the von Gräfe dilator.

Scleritis, therefore, is an extremely chronic disease, developing in middle and old age. Relapses are easy. Fundamentally, gout, tuberculosis, syphilis or rheumatism is causative.

**Prognosis** in the superficial form is fairly good, if we do not consider the long duration of the disease. In the deep form, blindness is a frequent sequela (pupillary atresia, secondary glaucoma, etc.).

**Therapy** is somewhat powerless. Atropin to full dilatation of the pupil, warm, moist compresses, wet packs, eventually subconjunctival injection. The nodes may be massaged with mercurial ointment.

Constitutional treatment, according to the nature of the fundamental affection, is very important. If increase of intraocular pressure develop, iridectomy is indicated.

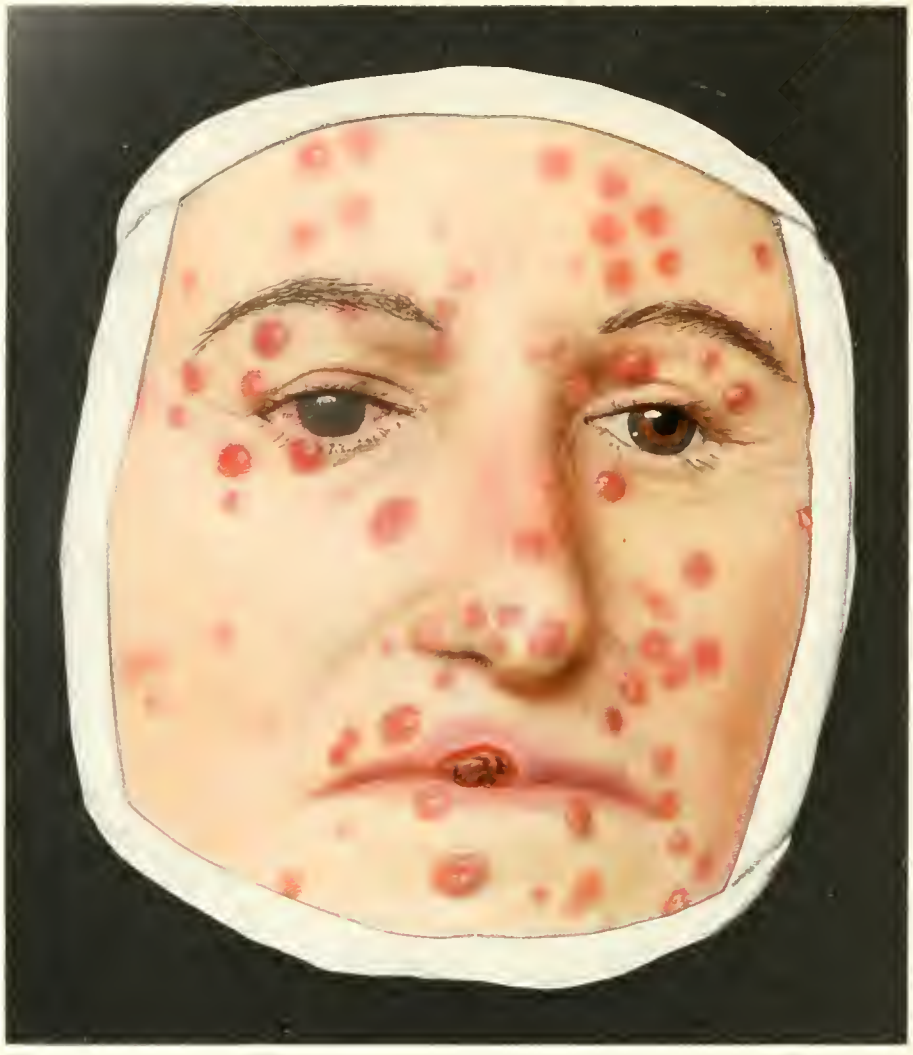

Fig. 64.
Iritis. Papulous Syphilite.

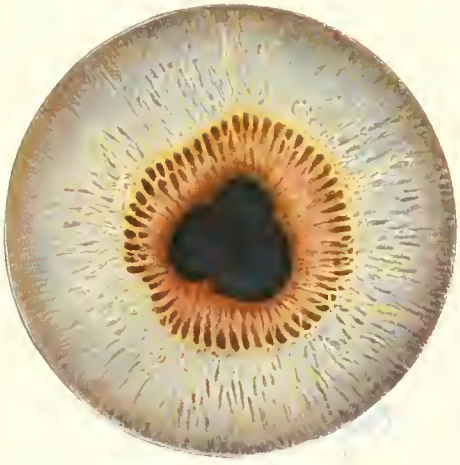

Fig. 65. Synechiae posteriores following Iritis.

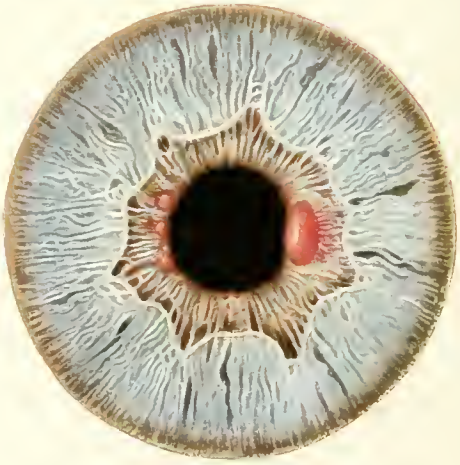

Fig. 66. Condylomata iridis.

# Iritis.

Plate XLVI., Figs. 64-66; Plate XLVII., Figs. 67-69.

Iritis is a common and serious lesion, and knowledge of it is essential to every practicing physician, for its non-recognition or improper treatment will lead, in a few days, to adhesions of the iridic margin which cannot be corrected, disabling the patient during his entire life, even if such neglect do not, as is common, finally lead to loss of the eye.

The symptoms of iritis are: (1) A *pericorneal or episcleral injection* (*vide* Plate XLVI., Fig. 64) composed of violet-red, deep blood-vessels, branching like the wisps of a broom, close to and surrounding the cornea like a ring. These vessels lie beneath the conjunctiva upon the sclera, and by their marginal branchings nourish the deep layers of the cornea and then, at the junction of cornea and sclera, turn inward and penetrate to the iridic attachment. In violent inflammations, not only is the red ring around the cornea present, but the entire eyeball is red. We must, however, verify that the redness augments as it approaches the corneal margin, whilst in simple conjunctival redness it decreases (*vide* Plate XXXVII., Fig. 54).

(2) *Alterations in the pupil.* Very early the pupils contract and exhibit a tardy reaction (because of cramp in the musc. sphincter iridis). If left in this contracted state, fibrinous exudates develop on the posterior surface and adhere to the anterior capsule of the lens (synechiæ posteriores), first at one point,

then at several, finally including the entire iris. The synechiæ are sometimes first seen after the instillation of atropin, when the free areas of the iridic margin respond with dilatation whilst the adherent areas are directed inwards like well-defined serrations (*vide* Plate XLVI., Fig. 65; also, Figs. 67-68).

Finally, in the still free, contracted pupil, surrounded by adhesions, a fibrinous deposit may be observed, entirely occluding the pupil: Occlusio pupillæ, due to an inflammatory membrane (*vide* Plate XLVII., Fig. 69).

(3) *Changes in iridic tissue.* The hyperemia of the iris soon leads to a change of color, so that the blue iris becomes a dirty green, the brown iris a rusty red.

In some few cases, characteristic nodules form on the surface of the iris. Those appearing on the pupillary margin, multiple, and, because of the rich vascular supply, having an orange-yellow tint, are to be considered as luetic papules or condylomata (*vide* Plate XLVI., Fig. 66).

The pale-gray tubercles, poor in blood-vessels, are mostly shown about the small iridic circumference or in the corner of the anterior chamber (Plate XLVII., Fig. 68).

Gummata may develop as large yellow nodes, usually isolated (Plate XLVII., Fig. 67).

(4) The *cornea* is often mistily opaque.

Subjectively, we have photophobia, lacrimation, and frequently violent pains, which may extend to the forehead and temples.

**Course of the disease.** Iritis may be either extremely insidious or else violent with much pain. It is always to be considered as a very serious lesion. Its course may extend over weeks or months, the period depending largely upon the treatment.

**Etiologically,** it should be thoroughly understood that we are dealing with a constitutional disease, for the idiopathic iritis of olden times does not exist.

Most often a new syphilitic infection, acquired some months previously, is etiologic, but it is also found in old cases. Next in frequency as an etiologic factor is tuberculosis, and the iritis is directly due to metastasis from some gland.

It is not a rare metastasis in gonorrhea, and may develop even after the urethral lesion has been cured.

As a rare phenomenon it may occur in various infectious diseases.

That a simple iritis may be caused by a violent cold is very dubitable.

Naturally, disease germs entering the eye through an abraded or injured surface may develop an iritis traumatica.

**Prognosis,** when correct treatment has been employed, is commonly good, and in the great majority of cases, the eye may be spared any injury, whilst by non-recognition of the trouble the eye may be seriously and permanently injured or lost within a few days.

**Therapy.** In iritic inflammation atropin is the greatest remedy; by its narrowing of the iris the blood supply is lessened, and it hinders the development of adhesions between the pupillary margin and the capsule of the lens (posterior synechiæ). With an inflamed iris, mydriasis is much more difficult than when the iris is normal. The action of the drug is accentuated if used warm and cocain added (thus dilating the lymph spaces and increasing the resorptive powers). Atropin (1%) is instilled 6-12 times at inter-

vals of a few minutes until the pupil is fully dilated, or it is seen that adhesions have already formed and hence, further dilatation is impossible. After mydriasis is obtained, the pains usually abate. Cold is poorly borne; warm compresses (chamomile or boric acid 2-4%) are, on the contrary, very helpful; eventually, warm, moist packs are indicated; where the pains persist, cupping of the temporal region may be resorted to. Thorough sweating (Schwitzkuren) often affords marked relief. If an annular posterior synechia exist, iridectomy should be done after subsidence of the inflammation, in order to avoid secondary glaucoma.

Above all else, the etiology of the iritis should be determined, and the indicated constitutional treatment pursued.

Fig. 67.
Gumma iridis. Synechiae posteriores

Fig. 68.
Tubercles of the Iris. Synechiae posteriores.

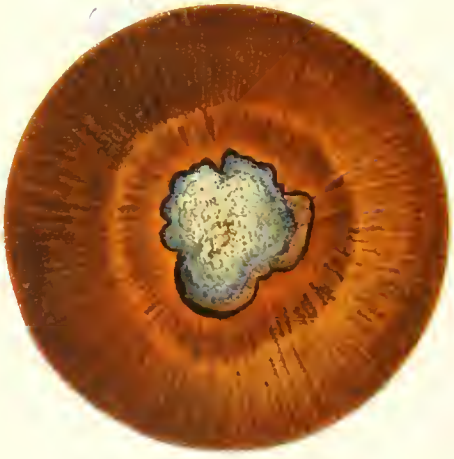

Fig. 69.
Occlusio pupillae
Membrana pupillaris inflammatoria.

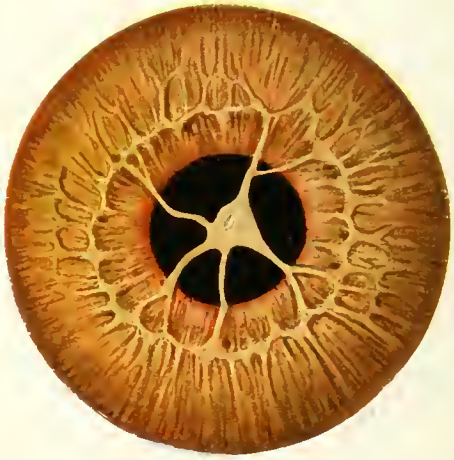

Fig. 70.
Membrana pupillaris perseverans.

# Congenital Anomalies of the Iris.

## I. Coloboma Iridis Congenitum.

Plate XLVIII., Fig. 71.

## II. Membrana Pupillaris Perseverans.

Plate XLVII., Fig. 70.

I.

*Coloboma iridis congenitum* may be subdivided into a typical and an atypical form. In the typical form, there is a split or fissure in the iris, inclining downwards and, sometimes, a little towards the nose. Its form resembles most that of a Gothic window, the apex extending either to the ciliary edge (coloboma totale), or the process stops before it reaches the line of the eyelashes (coloboma partiale). The coloboma, with its apex invariably rounded off, enters into the pupilla, and, in this area there are sometimes found, extending in a horizontal direction, cords or strings of connective tissue passing over the coloboma, (bridge coloboma, Brückenkolobom). An unpigmented band or cord extending downwards is termed pseudo-coloboma.

Of atypical colobomata there are numerous reports in the literature, the defects extending in all directions.

The congenital coloboma is the result of imperfect closure of the palpebral fissure *in utero*.

## II.

The *membrana pupillaris perseverans,* the remainder of fetal pupillary membrane, is not extremely rare. It is differentiated from inflammatory deposits upon the capsule of the lens by the fact that the threads or cords of tissue never have origin in the pupillary margin, but always upon the anterior surface of the iris, most commonly from the circulus arter. iridis minor.

In the fetal eye it is well known that the hyaloid artery passes forward through the central canal (canalis Cloqueti) of the vitreous. On reaching the posterior pole of the lens, it branches and forms a vascular network, the membrana capsularis, investing the entire posterior surface of the lens. These vascular branches pass over to the anterior surface of the lens and unite there with blood-vessels derived from the anterior surface of the iris (especially those from the circulus arteriosus irid. minor). In the pupillary region, a membrane thus developed, is called the membrana pupillaris.

Fragments of the pupillary membrane are not infrequently noted but it is extremely rare to find portions of the membrana capsularis on the posterior surface of the lens.

The normal pupillary membrane in the fetus is finely granular with oval nuclei irregularly distributed in it. The anterior surface is covered with an epithelium at first unimpaired and continuous, but later it becomes more and more defective. On the posterior surface, blood-vessels covered with an endothelium spread out. With retrogression of the disease, the epithelium disappears first, then the blood-vessels, and the membrane itself serves as endothelium for the iris.

The membrane, when it persists, is found to be a tough tissue, poor in nuclei, but with scattered pigment cells. Upon both the anterior and posterior surfaces, the layer of endothelium is, usually, imperfect. The filaments or threads directly penetrate the iridic tissue from whose structure they cannot be differentiated. The blood-vessels in it are almost invariably empty.

# Glaucoma.

Plate XLVIII., Fig. 72.

The essential nature of glaucoma (grüner Star) is the heightened pressure within the globe. It may develop rapidly (glaucoma acutum or inflammatorium) or very gradually (glaucoma chronicum or simplex).

The actual reason for the increase in pressure is not known. Anatomically, we usually find the angle of the anterior chamber where the humor is drained off through the spaces of Fontana into the canal of Schlemm, occluded by inflammatory adhesions.

True glaucoma generally exhibits prodromal symptoms. The patient, from time to time, has obscuration of vision, sees as through a fog, and lights used for illuminating purposes (candle, gas, etc.) are surrounded by colors, rainbow tints: stadium prodromale. These phenomena may be called forth by slight increase in pressure.

Earlier or later, more violent attacks occur: stadium evolutum. In acute glaucoma there is marked pericorneal injection, violent, even unbearable, pains. The cornea is mistily opaque, the anterior chamber much diminished in volume, the iris discolored, the pupil spontaneously enlarged, and vision, more or less noticeably impaired. The bulbus is distinctly hard to the touch.

In chronic cases all these phenomena of inflammation are absent, and, generally, the increase in pressure is not felt manually. The patient is conscient only of poor vision. Diagnosis is best and earliest as-

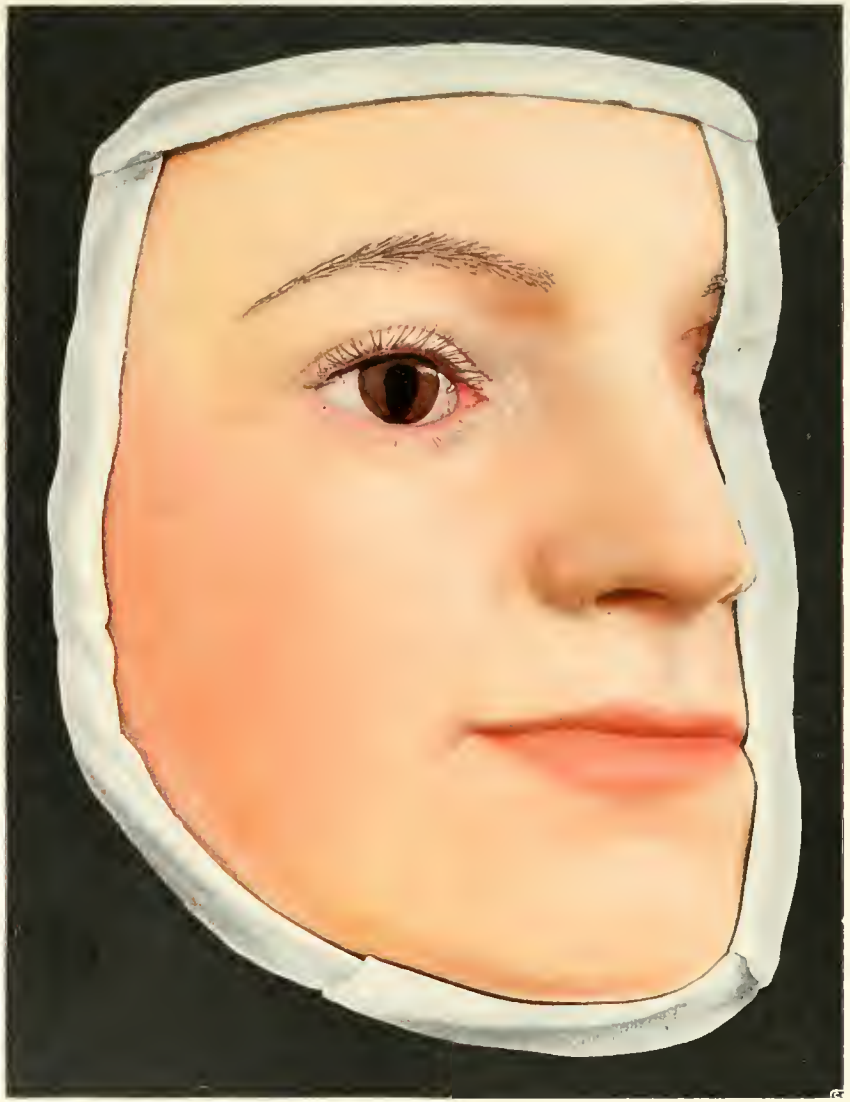

Fig. 71.
Coloboma iridis congenitum.

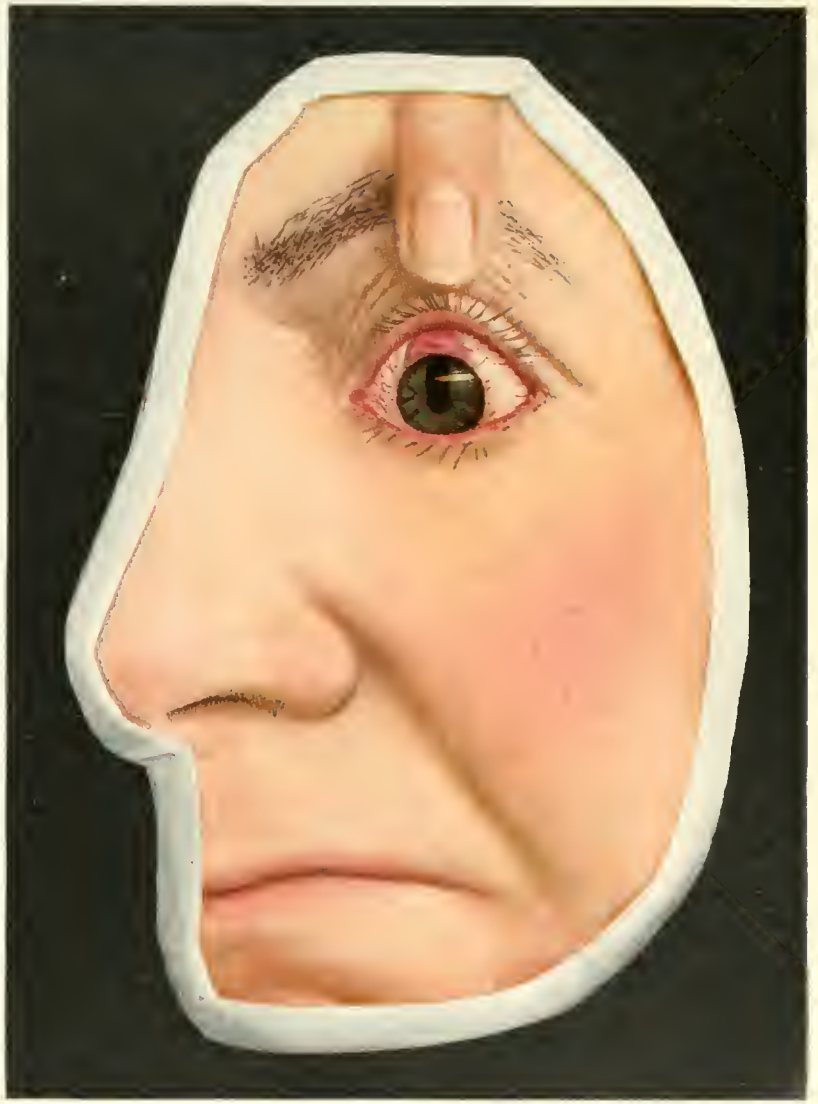

Fig. 72.
Coloboma iridis artificiale.
Glaucoma inflammatorium

certained by determining the limits of the field of vision, which starting from the periphery diminishes slowly but continuously by sectors, particularly on the nasal side. If this diminution be already noticeable, then the second sure symptom of chronic glaucoma is present, *i. e.* the ophthalmoscopically visible excavation of the papilla nervi optici.

Left undisturbed, the disease leads surely, sooner or later, to absolute, incurable blindness: glaucoma absolutum.

All the optic nerve fibres are broken at the margin of the deep, kettle-like excavation and become atrophied. Later, there almost invariably develops a total opacity of the lens.

**Therapy.** The miotics, physostigmin (=eserin) and pilocarpin are suitable remedies for reducing the pressure. They act as long as the iris is not atrophic; *i. e.* as long as it is capable of contraction. Their action, also, is not permanent, for with the subsidence of miosis, the heightened pressure returns, so that we have only palliative, not curative, remedies. Beware of atropin, for it augments intraocular pressure. If the diagnosis of glaucoma is absolutely sure, iridectomy should be done as soon as possible. Other authors recommend sclerotomy in its stead.

Generally, one may expect to save whatever vision is left by a well-executed, broad, peripheral iridectomy.

# Disturbances of Iridic Motility.

Plate XLIX., Fig. 73 (Tabes).

Disturbances of iridic motility are shown by the behavior of the pupil.

Normal pupils are, as a rule, equally large, whilst inequality (anisocoria) always indicates a pathologic condition. Either the pupil is continuously and permanently abnormal in regard to its contraction or dilatation (*e. g.* often and very early in cerebral syphilis), or else the condition changes so that one pupil suddenly becomes much dilated or contracted (Springende Pupillen, an early symptom of progressive paralysis).

Normally, the pupils contract equally when light is thrown into them, and it is a matter of indifference into which eye the light is cast, for the other eye reacts similarly (consensual reaction).

Above all else, we investigate the pupillary reaction from direct light and the convergence reaction of both eyes. If the pupils remain inactive in these two tests, there is an absolute pupillary immobility; if they do not react to light but respond to the convergence test, it is terméd a reflex immobility.

The case represented in Fig. 73 is that of a man, æt. 45, who, when 30 years of age, was infected with syphilis.

Anisocoria is present, the left pupil is strikingly contracted (spinal miosis) and, furthermore, it is clearly seen that the pupils are not circular, a condition very indicative of tabes and paralysis (entrundete Pupillen), not to be confused, however, with the

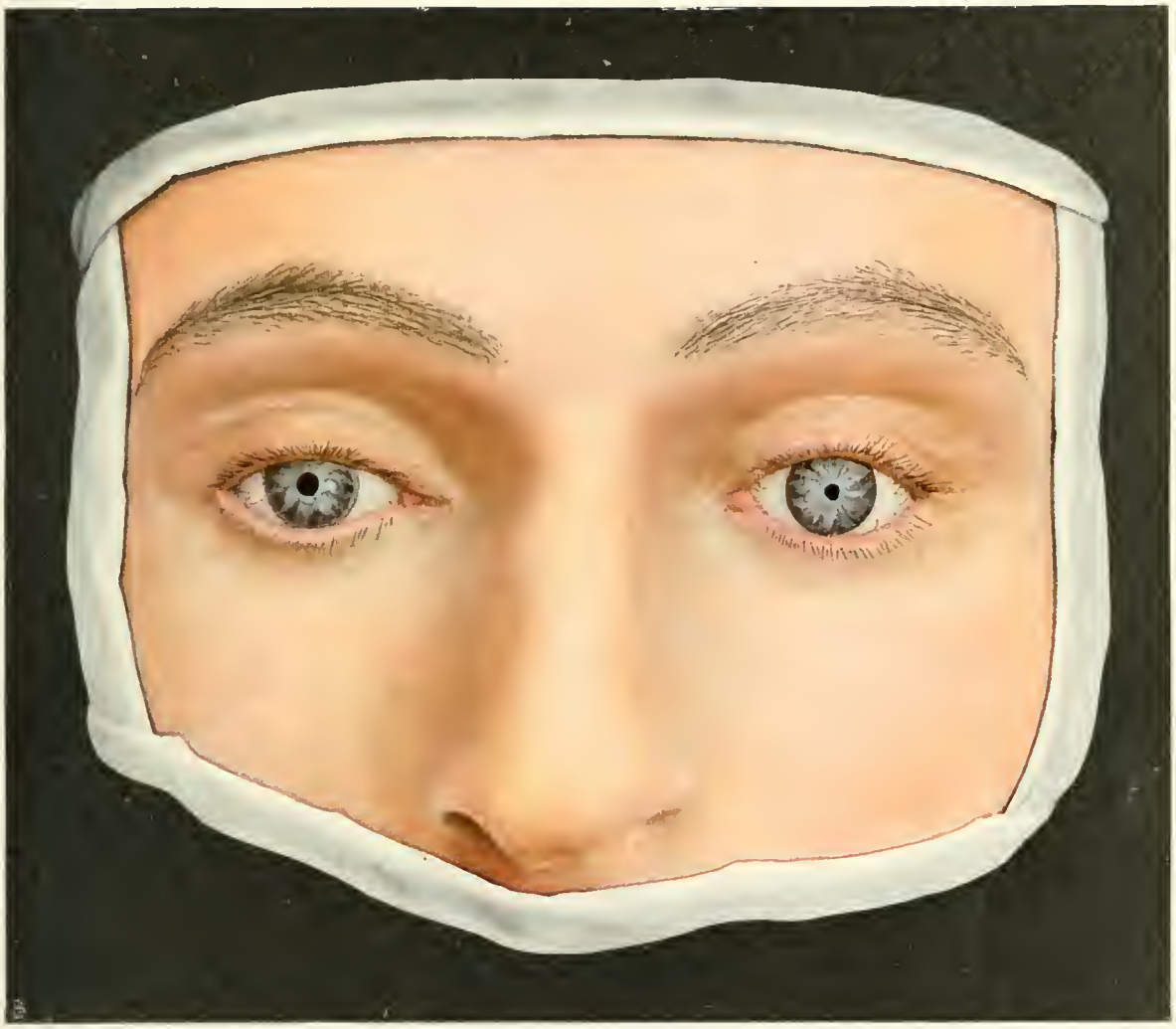

Fig. 73.
Anisocorid. Reaction of pupils in Tabes.

dentated pupil from synechiæ, adhesions due to inflammation.

There is also a reflex immobility of the pupil, *i. e.* both pupils remain unaffected by light but react to convergence.

The accomodation also is paretic, a fact which, primarily, led the patient to the oculist.

Vision and the field of vision are normal and hence there is no atrophia nervi optici tabetica incipiens present.

The patient, with the exception of a little rheumatism, considers himself perfectly well. Lancinating pains, however, with anesthetic areas in the lower limbs, absence of knee-jerk, and the presence of Romberg's sign confirm the diagnosis of tabes dorsalis already arrived at by the oculist.

# Cataract (Grauer Star).

Plate L., Fig. 74; Plate LI., Fig. 75; Plate LII., Figs. 76-79.

Morbid alteration of the lenticular tissue is usually due to disturbance of nutrition, which exhibits itself as opacity of the lens: cataract (κατα and ῥήγνυμι, to fall down, descend).

Cataracts vary much in regard to species and composition, and may, therefore, be classified from various points of view, *viz.*:

I. *Progressive* (*e. g.* cataracta senilis, which continuously augments with age, although with various degrees of rapidity) and *stationary* (*e. g.* cataracta zonularis, which remains unchanged for years or even a lifetime).

II. *According to maturity.* A cataract is said to be mature when the lens is completely opaque, even to the capsule and into the periphery. In accordance with this, we differentiate as progressive forms: Cataracta incipiens, nondum, fere matura, hypermatura, the last indicating a retrogressive process following maturity, and characterized by shriveling, proliferation of capsular endothelium, calcification, etc.

III. *Simplex or complicata.* A better nomenclature would be "primary" or "secondary," but the latter is already used to designate another form of cataract, *i. e.* the more or less thick membrane left after extraction of the lens, or which is formed anew from the residual capsule, endothelium, and fragments of the lens not extracted. Cataracta simplex is present when

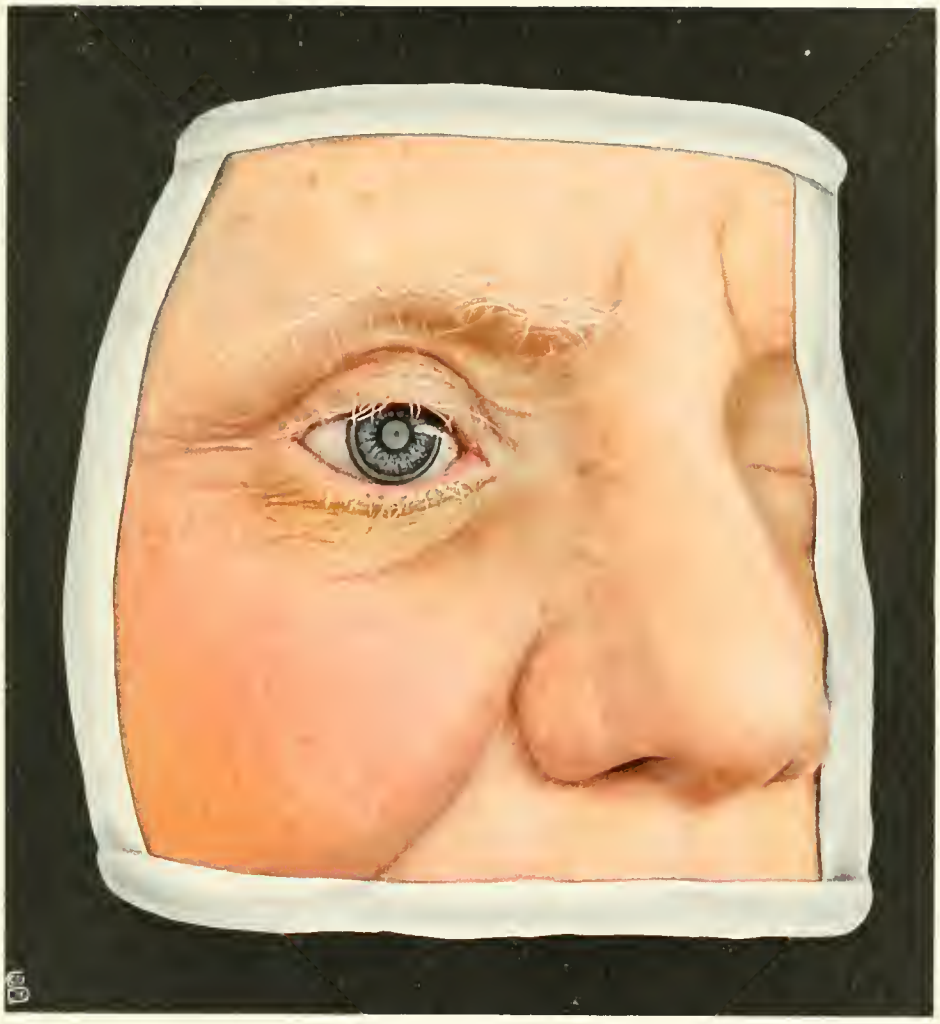

Fig. 74.
Arcus senilis corneae. Cataracta senilis matura.

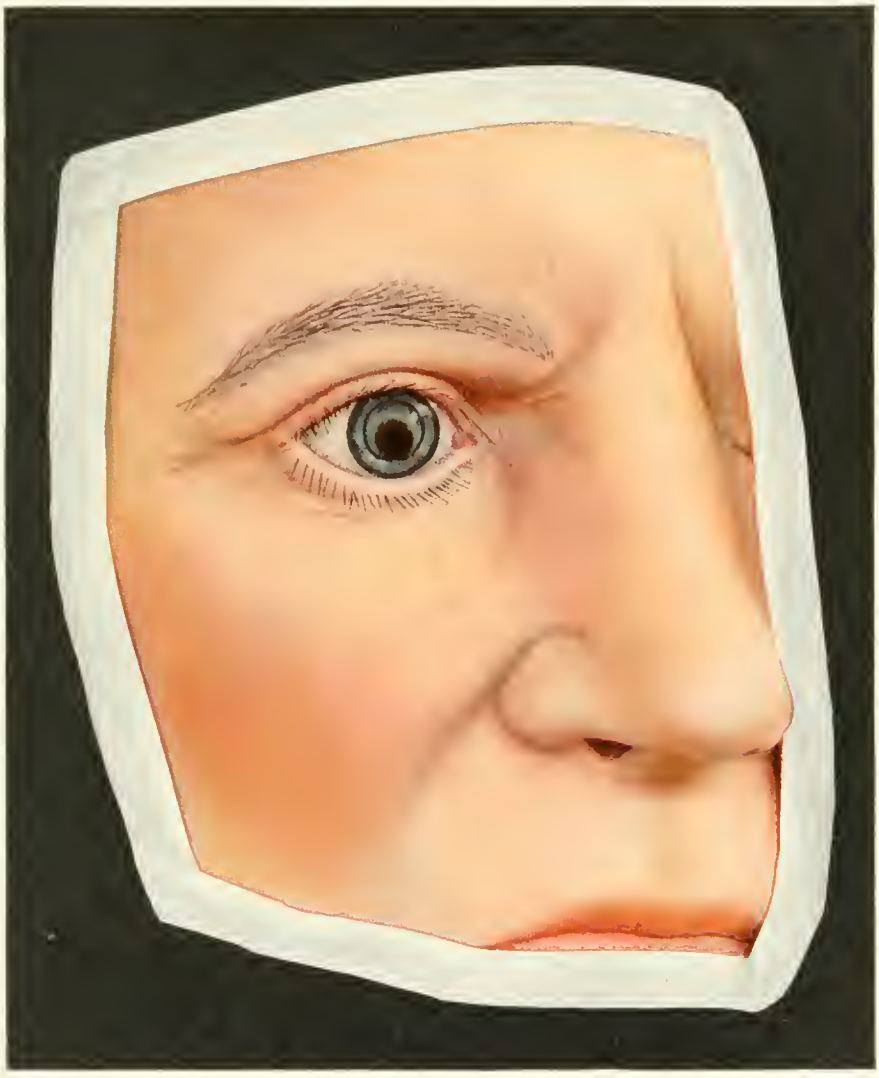

Fig. 75.
Cataracta morgagniana.

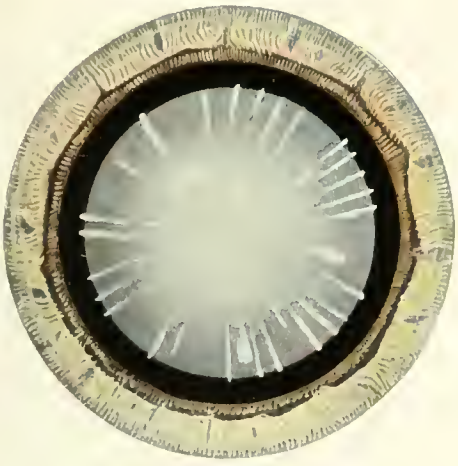

Fig. 76.
Cataracta zonularis

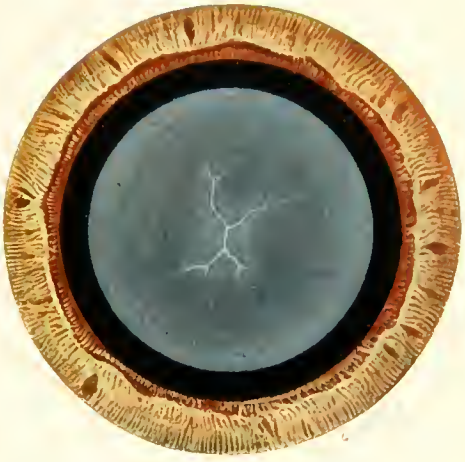

Fig. 77.
Cataracta zonularis with Cataracta stellata

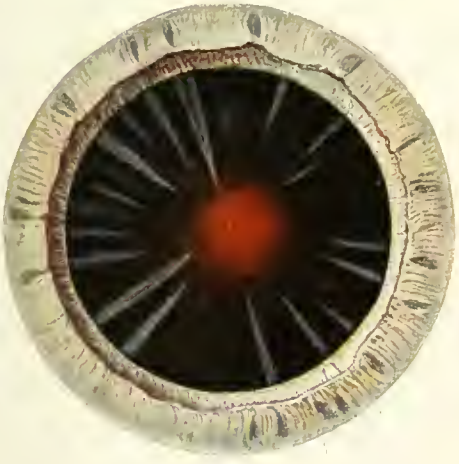

Fig 78.
Cataracta senilis incipiens.

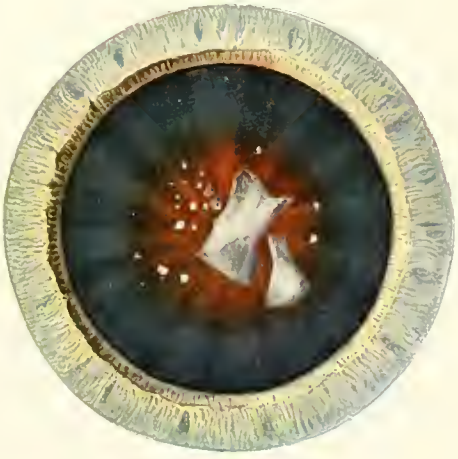

Fig. 79.
Cataracta capsularis.

the lens alone is affected but is yet able to perceive light, the eye being otherwise sound. We speak of cataracta complicata when, to an internal blindness, there is added an opacity of the lens. Both forms may, externally, look alike, but clinically they are easily differentiated by the tests for light and projection.

IV. *According to the age of the patient*: Cataracta congenita, mollis or juvenalis, dura or senilis. The congenital cataract or that found in youth is not always amenable to strict classification, and offers scarcely any pathologic or anatomic deviations. We have no precise knowledge in many cataract forms whether they were congenital or first developed in the early years of life, and we are often dependent upon anamnesis only. Contrarily, there is great difference between the soft and hard forms. In youth, the lens is of a soft and uniform consistency, and when removed with its capsule endeavors to assume a globular form. In the course of years the central portions gradually become denser and harder, and usually when the patient attains the age of 30 there is present a firm, unyielding, unchangeable nucleus or kernel which does not react to contact with the aqueous humor. After that age, therefore, we consider the cataracta dura as important from the viewpoint of operative ophthalmology.

V. *According to the form.* Cataracta zonularis is the most frequent and characteristic form in youth. In this zonular or lamellar form, a cloudy or opaque stratum develops between the center, which remains transparent, and the cortex (*vide* Plate LII., Fig. 77). Sometimes from this, small, densely opaque projections push peripherally into the transparent zone (Plate LII., Fig. 76). From cataracta zonularis, a number of other forms may develop, for example:

If the opacity extends through the center, we have a cataracta centralis (not nuclearis, since no nucleus is present before the age of 30).

If the periphery also is likewise involved, we have a cataracta totalis. These three forms may pass into or develop from one another, and the cataracta totalis may undergo the following modifications:

Its contents may reach such a degree of softening that they become thin and milky, cataracta lactea, or clear so as to resemble a cyst, cataracta cystica. Later, not only the subcapsular stratum but the contents may calcify, become hard and chalky-white, cataracta calcarea. If the watery contents be early resorbed so that there is left only a thin wall, composed of capsule and some epithelial proliferations, we have a cataracta membranacea, or if the epithelial cells beneath the capsule calcify, thus forming a thin, stiff, chalky-white stratum, we have cataracta papyracea. If, however, the capsule is not reduced to a papyracous layer, but remains distended after resorption of its contents, it is called cataracta aridosilicata.

Cataracta fusiformis (spindle-shaped cataract) or axialis is a congenital and rare form, having a spindle-shaped opacity in the axis of the lens, joining the anterior and posterior poles.

Cataracta capsularis. There is no actual opacity of the capsule. The opacity is in the anterior surface of the lens beneath the capsule, and, after calcification, is white in color (*vide* Plate LII., Fig. 79). If such an opacity is located like a point or dot just at the anterior or posterior pole, we have, respectively, a cataracta polaris ant. or post. The former may project like a pyramid or cone into the anterior chamber, cataracta pyramidalis.

In cataracta stellata the cement substance of the lenticular fibers is clouded so that the star becomes

visible on the anterior or posterior surface. In Fig. 77 (Plate LII.) this star figure is seen at the anterior pole of a cataracta zonularis.

Cataracts appearing in middle life often consist of gray points, cataracta punctata, or stripes, cataracta striata.

VI. *According to origin,* we have: Cataracta senilis (corticalis or nuclearis), beginning, usually, with spoke-like opacities lying between the nucleus and cortex, and which, wedge-like, push from periphery to center, thus resembling spokes in a wheel (*vide* Plate LII., Fig. 78). This grayish clouding of the cortex, which later becomes more homogeneous (Plate L., Fig. 74), is to be distinguished from an opacity of the nucleus, which is more of a brownish-red in color (Fig. 78-79).

Cataracta nigra is a peculiar form of senile cataract, where the pupils appear black although the lens is opaque. It is due to extreme sclerosis of the nucleus.

Cataracta morgagniana is a senile form where the cortex has liquefied so that the brown nucleus floats about in it. When the patient stands, the nucleus sinks (*vide* Plate LI., Fig. 75).

Other forms are cataracta diabetica, nephritica, traumatica. Forms due to some general disease are called constitutional cataracts, the best example of which is the cataracta diabetica, also the nephritica, and, according to many authors, the senilis. Poisonings may likewise cause cataract formation, *e. g.* from ergotin. The ergotin cataract is similar to the experimentally developed naphthalin, sugar, and salt cataracts.

If, moreover, a cataract has become adherent to the iris, an iritis with the formation of posterior synechiæ must have preceded it, and it is called cataracta accreta.

# Dermoids.

Plate LIII., Figs. 80-81.

Dermoids are congenital, cystoid tumors, whose walls are dermal in structure, although the tumors are found where, normally, dermal elements are absent.

In the eye they are found especially in two places: (1) At the corneo-scleral junction, and (2) under the skin on the bony margin of the orbit. In rare cases they extend from these sites farther into the orbit, thus interfering more or less with the eyeball.

(1) The dermoid tumors of the corneo-scleral junction are dense, of a porcelain-white or dull-rose color, and most often found at the external margin of the cornea between the muscle-insertions. They are but little elevated above the surface of the bulbus, but are deeply and immovably imbedded, partly in the corneal substance, partly in that of conjunctiva and sclera. They are always congenital, and so characteristic as to be difficultly confused with other tumors.

Microscopically, the structure is like that of a piece of skin: an epiderm of several strata, and below it a connective tissue stroma with sebaceous glands and hair follicles. The superficies is often covered with a fine down. Von Duyse attributes their development to adhesions betwixt amnion and the surface of the eyeball, whilst Remak considers them fetal invaginations of ectoderm.

Dermoids in children are usually small, on an average about the size of a lentil, but in later life they may

Fig. 80.
Dermoidcyst of the Cornea-scleral margin.

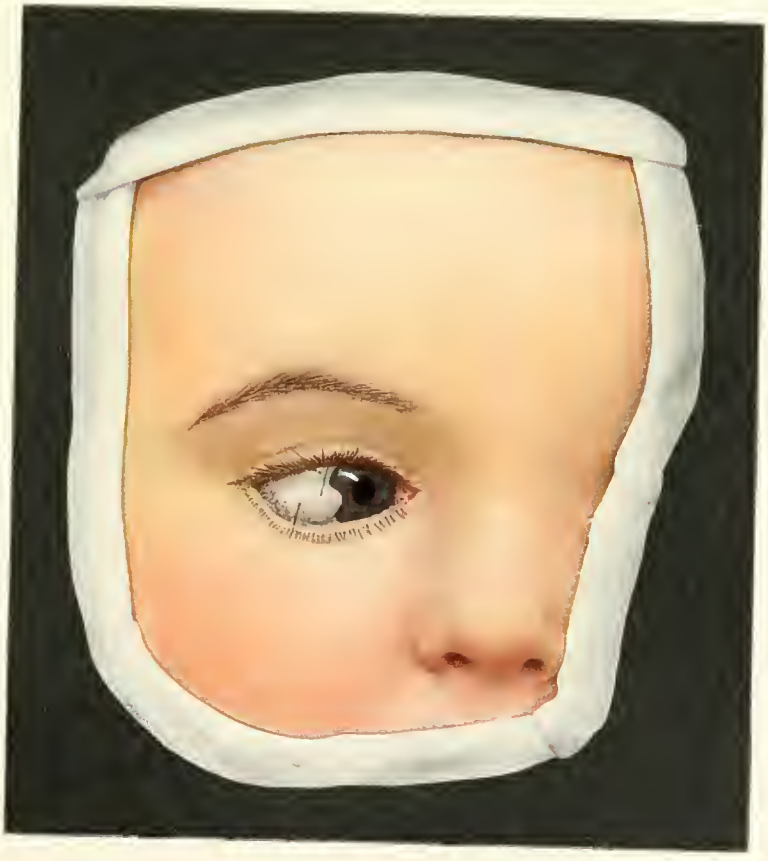

Fig. 81.
Dermoidcyst of the orbital rim.

begin to grow. Because of this and the ugly disfigurement of the eye, their early removal is to be commended. With most of these dermoids, the bulbus, as such, remains intact congenitally and is well formed. But a second, though rare, group show, in varying degree, a congenital interference of the dermoid with the development of the eyeball.

Schmidt-Rimpler described such a dermoid, the lens having been dislocated. On the cornea of a calf's eye was a piece of skin with hair, which began on the inner margin of the cornea, covering the greater part thereof. The iris had adhered to the corneal rudiment, and there was no anterior chamber. The lens projected through the pupil into the dermoid, thus being truncated.

A still more interesting case is from von Gräfe's clinic. The tumor was congenital, and during the eight months of the child's life had nearly doubled in size. It was divided into two large portions. The entire cornea, excepting a narrow border, was covered with an ordinary dermoid which was connected by a short stem with a dermoid of cherry-size projecting out of the palpebral fissure. The tumor was covered with cutis on which were a few hairs. Apparently, there was no lens in the globe. Microscopically, the dermoid showed cutis with hair, sebaceous glands, isolated papillæ, hair follicles. The cornea was lacking, its place being taken up by connective tissue, to which the iris, recognizable by a thick stratum of pigment, had grown.

Bernheimer describes a child, æt. 6 months, with two cherry-sized tumors in the right eye, which almost touched one another, keeping the lids apart. Later, they increased in size, entirely covering the cornea. Anatomically, there was a total corneal staphyloma with iridic proliferation.

In a case of Manfredi the well-formed orbit contained, not an eye, but a sphere the size of a pea. The abnormality increased somewhat with the growth of the child. The eyeball stump was covered anteriorly with a white, hairy skin. An excised portion showed the histologic structure of a dermoid.

Wagenmann examined a tumor from the orbit of a new-born child. Posteriorly it was joined to the orbit by a stem, the size of a quill. This stem was cut through and the orbit of the otherwise healthy and well-formed infant was precisely like that resulting from enucleation. The cavity was covered with a mucous membrane. On section, the tumor was found to contain a piece of bone and a rudimentary eyeball. A cross-section showed the tumor covered by a cutis about 1 mm. thick, with fine, down-like hairs.

II. Dermoid cysts of the skin of the lids or of their subcutaneous tissues are almost always located exteriorly on the orbital edge. They are not very infrequent, usually as large as a pea or plum-pit, though they may later increase in size. Commonly they are firmly adherent to the periosteum of the orbital margin. These, too, are congenital cysts with firm walls, and the pap-like contents are composed of desquamated, cornified cells and detritus, sometimes fat and hairs.

The tumors are to be considered as due to a stratum of germinal epithelium deposited on the areas affected.

It is well known also, that such dermoid cysts may be found deeper in the orbit.

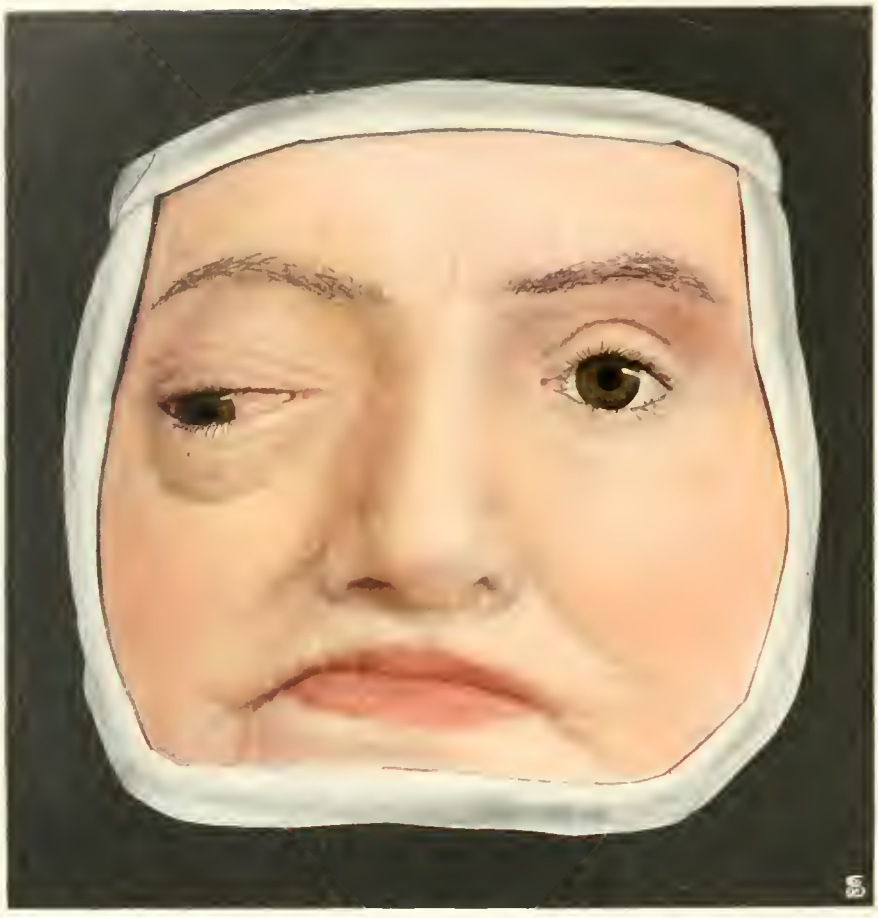

Fig. 82. Exophthalmus caused by Tumor retrobulbaris.

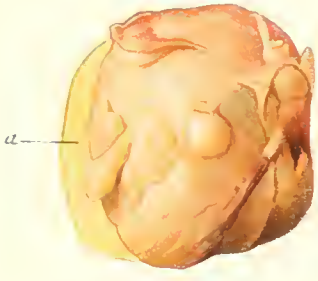


Fig. 83.
The Tumor after Exstirpation.

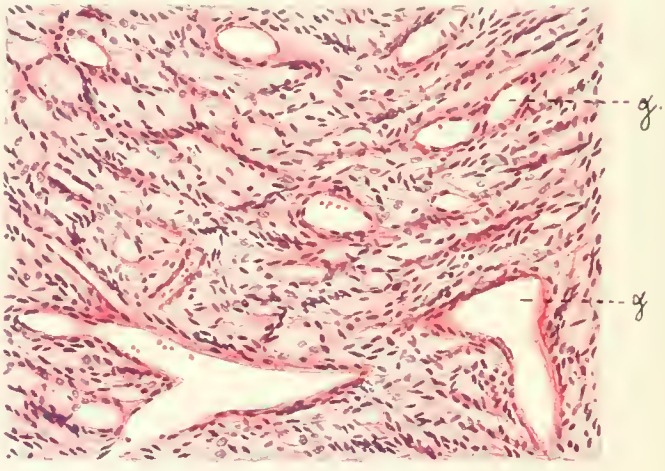


Fig. 84. Microscopic section of the tumor.

# Exophthalmus.

Plate LIV., Figs. 82, 83, 84.

By exophthalmus we understand a remarkable condition where the eyeball is, more or less, thrust forward out of its orbit. Exophthalmus is not to be confused with a condition where the eyeball is enlarged, and hence projects somewhat from the orbit, but still remains in its place.

Fig. 82 is the case of a woman, æt. 60, in whom the right bulbus in course of 2 years gradually, painlessly and without inflammatory symptoms advanced outward, forward and downwards.

In this case, the first species of exophthalmus, the inflammatory, could be excluded, for it develops suddenly, as a rule, or rapidly from exudates, often from collection of pus, or follows inflammation (periostitis, ostitis) of structures posterior to the eyeball. Frequently it follows the outbreak of pus from some cavity adjacent to the orbit. The lids become very edematous, swollen and red, chemosis sets in, etc. In our case there were only a few small blood-vessels of the conjunctiva bulbi dilated.

Nor were we dealing with a pulsating exophthalmus, which is usually caused by an aneurysma arterio-venosum due to a rupture of the carotid in the sinus cavernosum. In such case, the palpating hand distinctly feels a pulsation of the bulbus or the pulsation may be seen with the naked eye.

A non-inflammatory, simple exophthalmus is found

(a) in morbus Basedowü, (b) with retrobulbar tumors. The first possibility was easily excluded in this case.

We had to do, then, with a retrobulbar tumor, and, because of its very slow growth, with a relatively benign one.

Two circumstances permitted the elimination of a tumor of the optic nerve, which is not rare. In the first place such a tumor commonly drives the bulbus directly forwards, and secondly, vision was still fairly good, whilst in optic nerve tumors it is soon lost from compression of the optic fibers. (Moreover, the optic nerve lies behind the bulb in the form of the letter S, and hence, when pulled upon by an exophthalmus, is able to stretch or yield without danger.)

Nor were we dealing with one of the relatively more frequent tumors of the lacrimal gland, which would have pushed the eyeball downward and toward the nose.

The eyeball was quite movable; hence, the tumor was not adherent, and probably did not rise much above the adjacent tissue (*e. g.* the muscles). The nose and all cavities near the orbit were free, so that the tumor probably had origin in the orbit, and had remained there. Pressing inward with the finger on the upper lid between bone and eyeball, two hard nodes were encountered. Hence, cysts, entozoa (echinococcus, cysticercus) and lipomata, which are softer, were likewise eliminable.

We thus arrived at the exact diagnosis of a hard, probably capsulated, fibroma, whose site and size were just as accurately demonstrable.

**Therapy.** Removal of the tumor was indicated by the slow but steady growth. As soon as an exophthalmus attains a size which prevents closure of the lids, the situation becomes very tormenting.

As operative measures were discussed: The clearing out of the whole orbit, thus sacrificing the eyeball; the anterior removal of the growth, saving the eyeball; removal by the temporal route (Krönlein's method), saving the eyeball.

Since the tumor was indubitably benign and encapsulated, the first operation was not indicated; further, as its site was on the nasal side, the Krönlein procedure was abandoned.

After finding the tendon of the musc. rectus int. bulbi, which was caught up and cut, the bulb was drawn down and outwards with dull tenacula, and a hard tumor, the size of a small hen's egg, and attached to the deeper tissues only by a thin stem, was shelled out (Fig. 83), Microscopically, it proved to be a dense connective tissue growth, rather rich in cells (Fig. 84).

The eyeball was replaced and the muscle tendon sutured. There has been no return of the tumor, and the eyeball is in good condition.

# Index

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1909
Greeff, Richard
Atlas of external diseases of the eye